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A SCIENTIFIC CORNER

WHERE OPTICAL PROBLEMS ARE DISCUSSED AND
THE SOLUTIONS, AS SOLVED BY EXPERIENCE, INDICATED!

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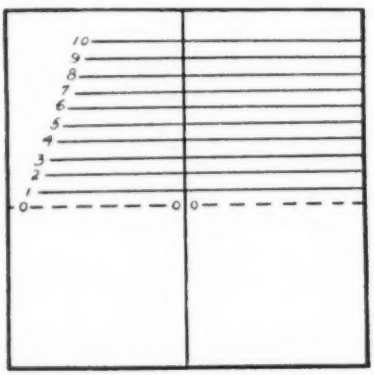
PRISM MEASUREMENT

In the April Scientific Corner, we presented the calipering method of determining prism power. This method, as we stated, is best applied to plano prisms, but may be applied to any prismatic lenses by first calculating the thickness difference caused by the correction—a tedious procedure. A very simple method of determining the prism power of any lens is by means of a calibrated scale.

This method is based on the universally accepted definition of a prism diopter by C. F. Prentice in 1886. Agreeable to this definition a one diopter prism produces a deviation of one centimeter at a distance of one meter. Therefore, at a distance of one meter a five diopter prism would produce a deviation of five centimeters and so on.



To use this principle for measuring prism power, it is necessary to have a scale hung on a wall at a known distance from the prism to be measured. We suggest using an 18 inch ruler, which is easily obtained, as a means of obtaining a fixed distance. One end of the ruler is placed below the zero mark on the chart, see cut, and the prism is held on the opposite end of the ruler with the apex of the prism down. The line on the chart corresponding to the power of the prism will fall on the zero line.

To figure the separation of the lines on the chart is a simple problem in ratio. If an 18 inch ruler is to be used as the distance from prism to scale, the separation of the lines must be 4.572 millimeters. 18 inches equal 457.2 millimeters, therefore, 1000 is to 10 as 457.2 is to x. x equals 4.572 millimeters. If a 12 inch ruler is used, the distance between the lines must be 3.048 millimeters. At two feet the lines on the scale would be 6.096 millimeters apart.

"IF IT'S A LENS PROBLEM, LET'S LOOK AT IT TOGETHER"

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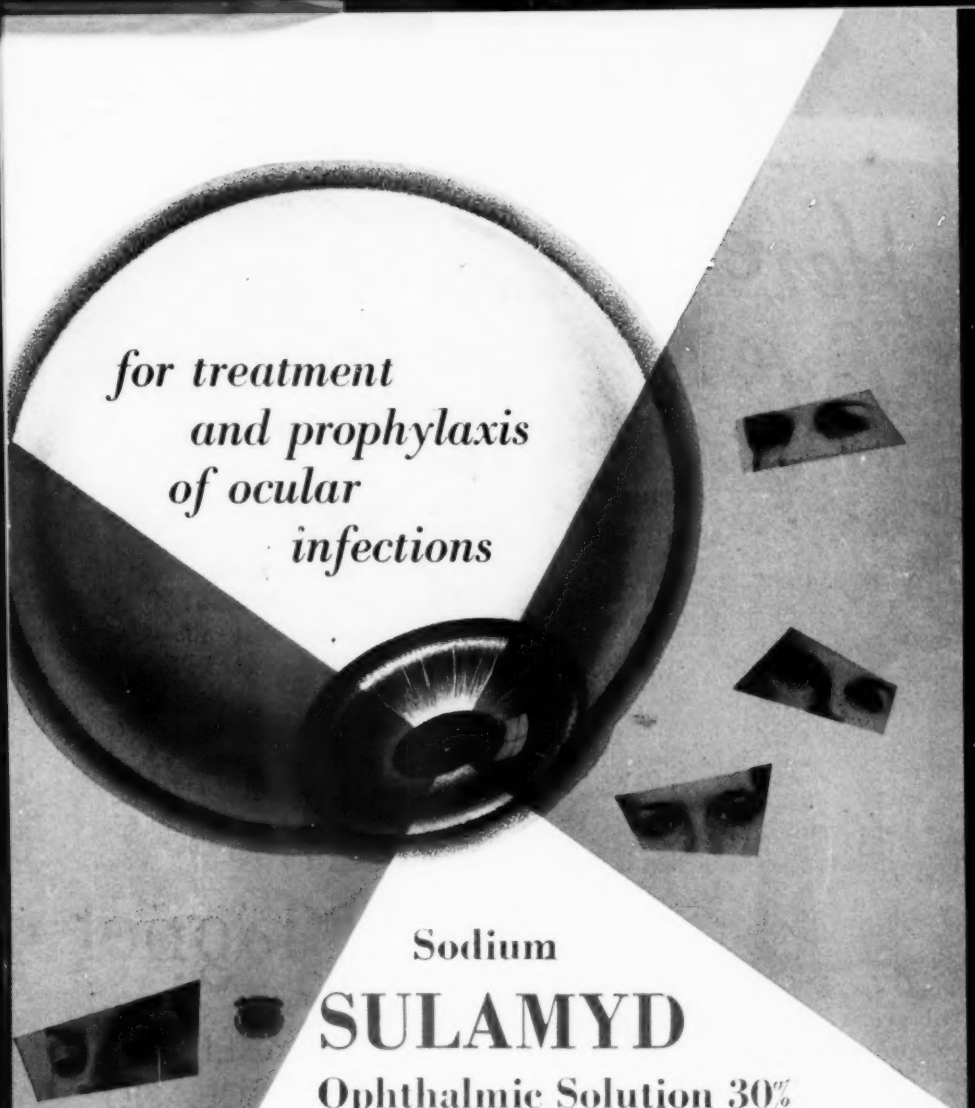

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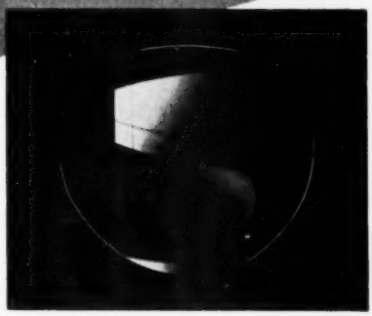

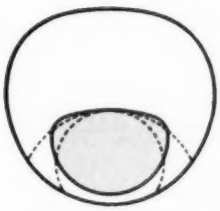

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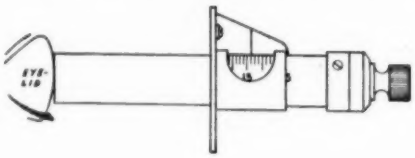

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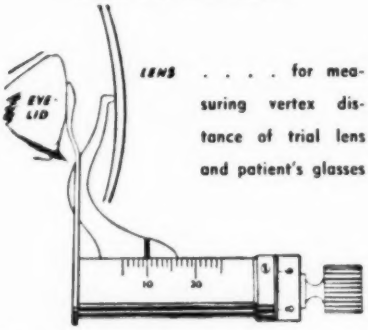

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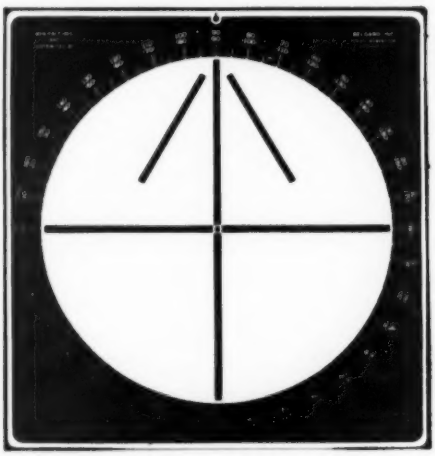

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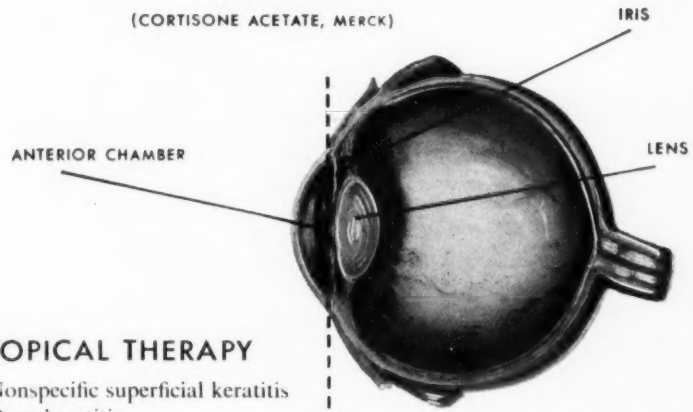

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1. Fine, M., and Goodwin, R. C.: *A.M.A. Arch. Ophth.* 47:787-797, June 1952.

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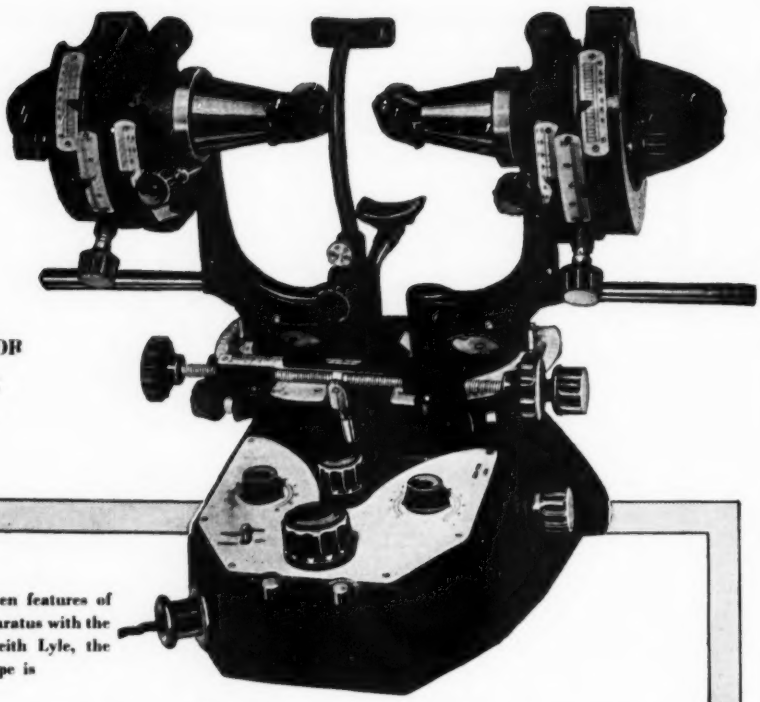

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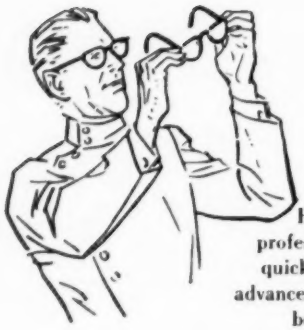

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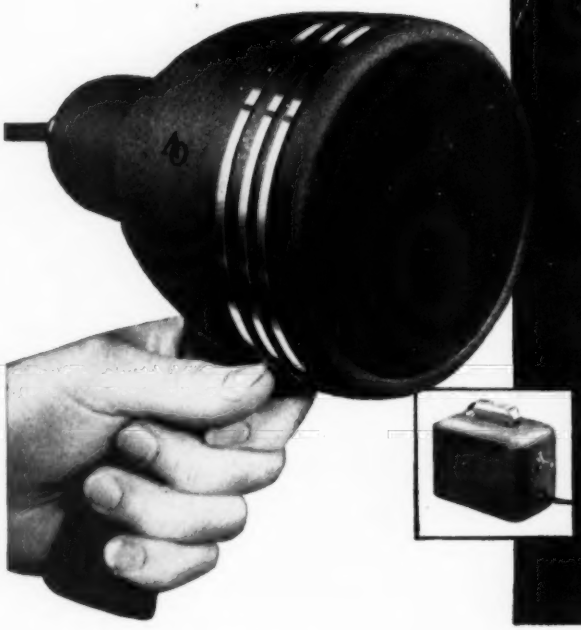

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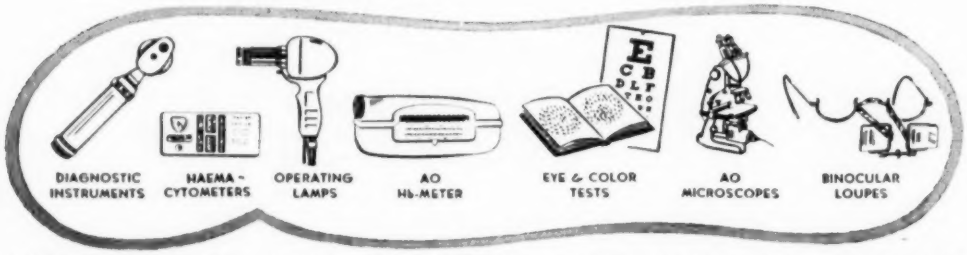

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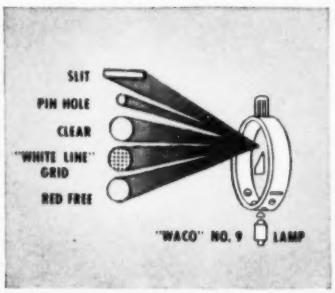

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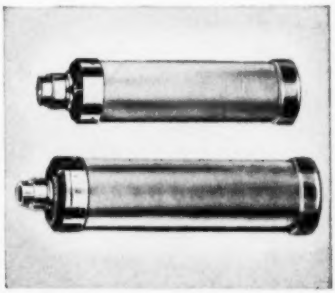

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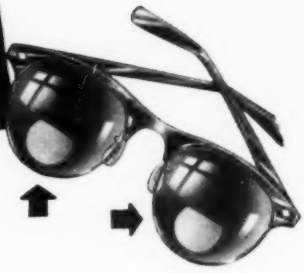

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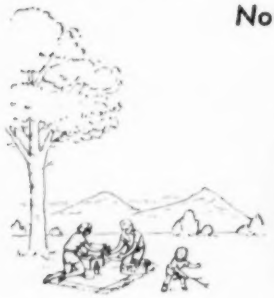

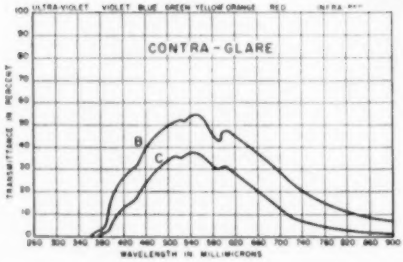

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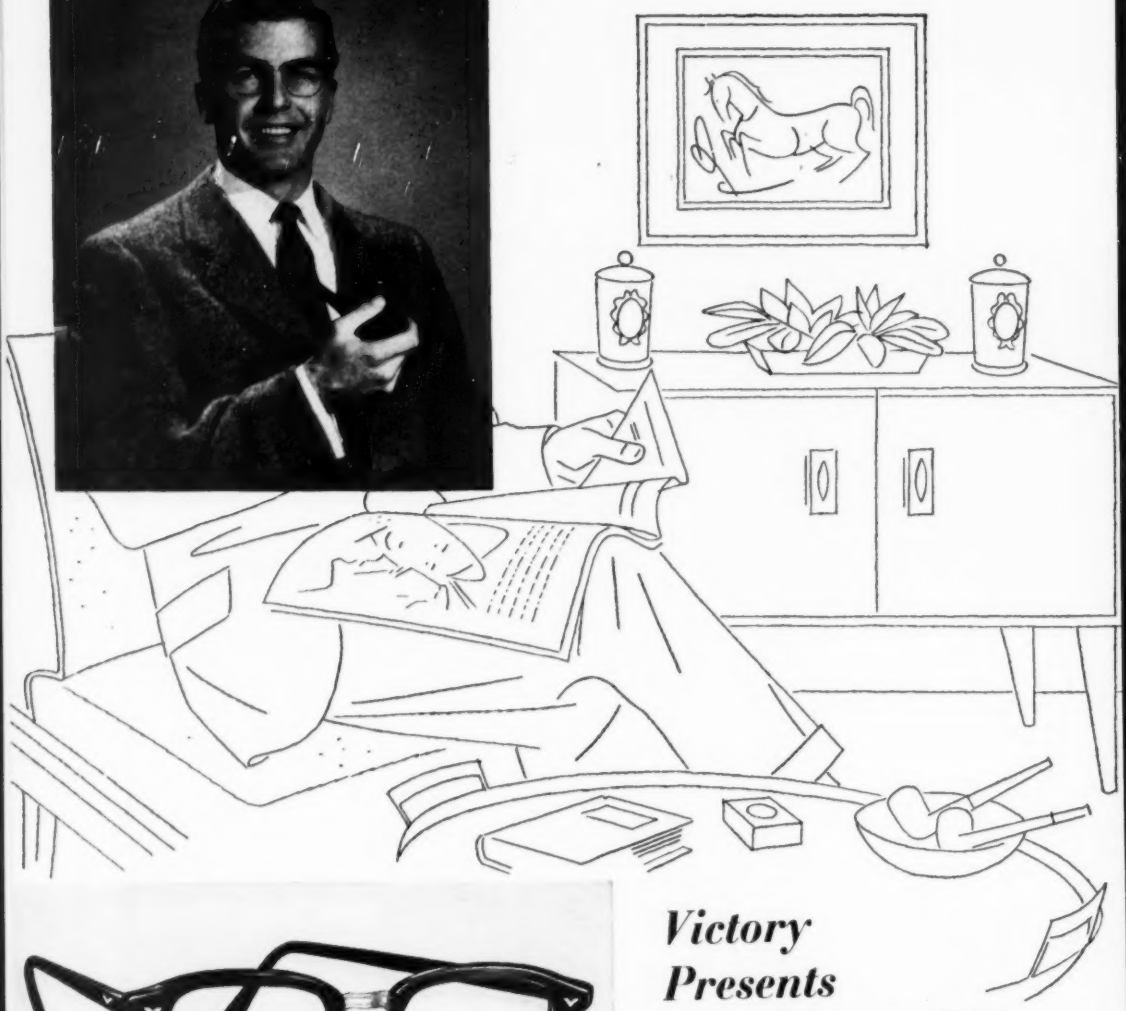

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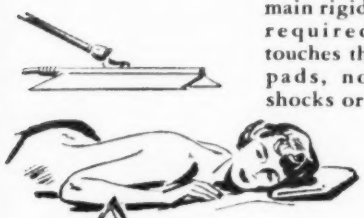

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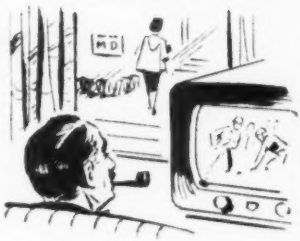

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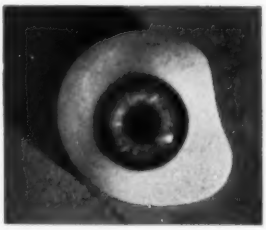

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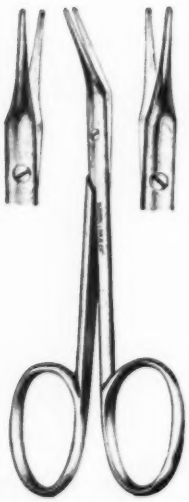

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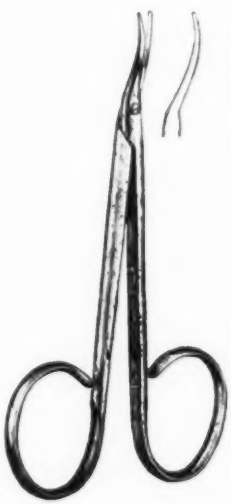

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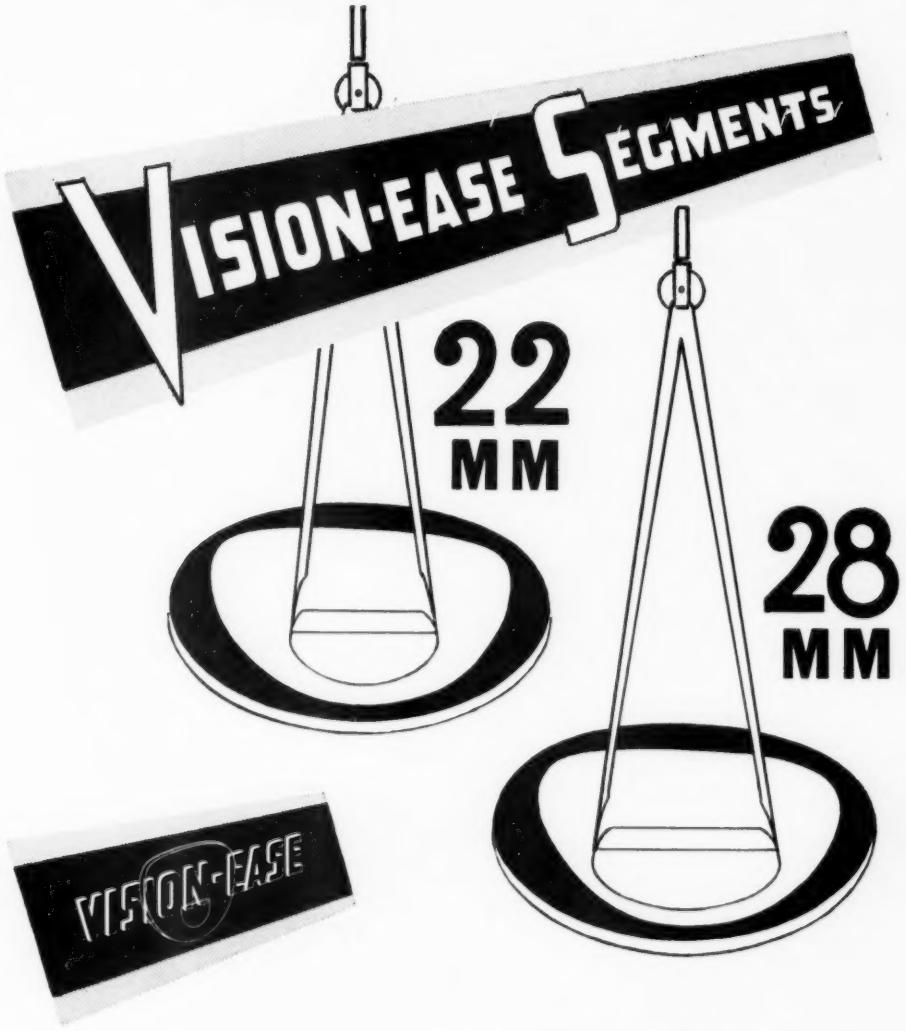

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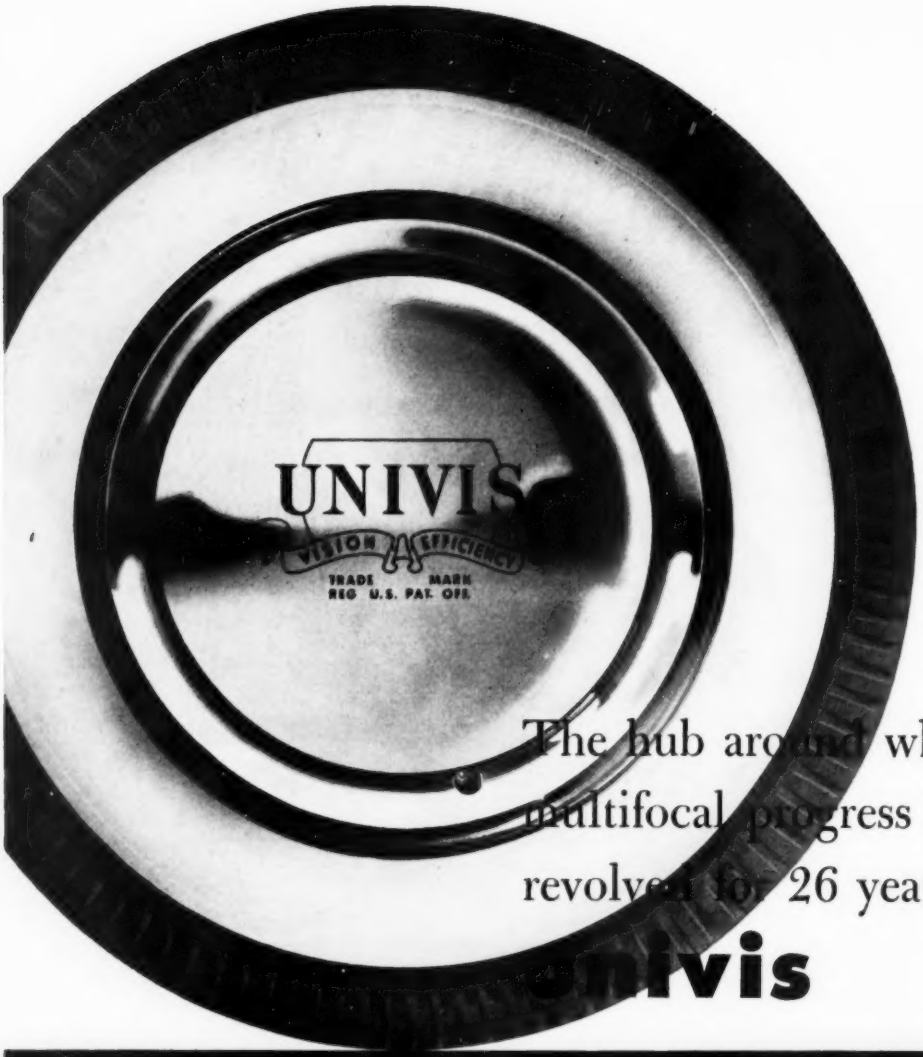

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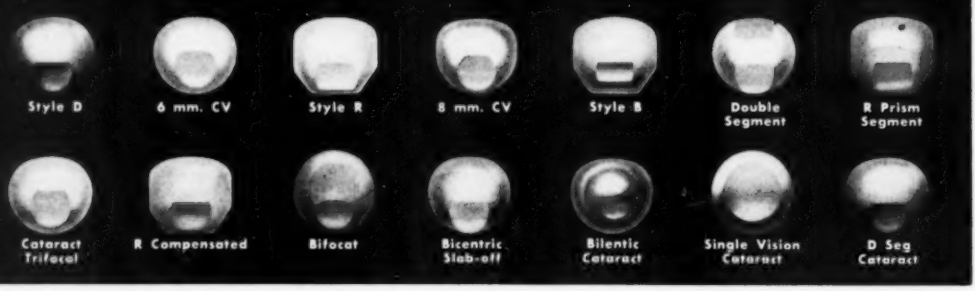

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VOLUME 49 JUNE 1953 NUMBER 6

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GENICULOCALCARINE INJURIES IN WAR CASUALTIES

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AND
CAPTAIN LLOYD S. DURKIN
MEDICAL CORPS, UNITED STATES ARMY

IT IS UNDERSTANDABLE that reports of traumatic visual field defects should appear during times of world stress. It is intended in this report to show the variety of visual field lesions encountered in the current conflict.

DIFFERENTIAL SIGNS IN LOCALIZATION

Congruity.—It is well known that the most important single point of differentiation between homonymous hemianopsias due to anterior lesions of the visual pathway (optic tracts and anterior radiations) and those due to posterior lesions (posterior radiations and occipital cortex) is the congruity of the visual field patterns of the two eyes.

It appears that visual fibers from corresponding points of the two retinas do not become closely allied until they have passed an appreciable distance beyond the chiasm. This is so despite the fact that corresponding fibers are projected onto identical areas of the visual cortex.

Lesions of the optic tracts produce homonymous hemianopsias in which the patterns of the two fields may be very dissimilar. Pure tract lesions are relatively uncommon. The optic tracts are not long, and lesions often involve the chiasm anteriorly.

In the anterior part of the optic radiation the fibers are only partly sorted; hence congruity is not quite complete. The fields become identical as the posterior portion of the radiation and the occipital cortex are reached. The congruity in all the cases to be reported is to be noted.

It is important to bear in mind that incongruity may be more apparent than real. The projection of the unilateral temporal crescent causes certain dissimilarities. For example, the temporal portion of the right visual field extends outward 90 degrees or more, but the nasal portion of the left visual field normally extends about 60 degrees to the right. Thus, congruity implies similarity of all portions of the fields except the temporal crescents.

Sparing of the Macula.—Another sign, though of less value, is sparing of the macula. Volumes have been written on the mechanism involved; suffice it to say that in many cases of occlusive vascular disease of the occipital cortex sparing due to an overlapping blood supply at the two occipital poles has been demonstrated. These vessels are branches of both the middle and the posterior cerebral arteries.

From the Departments of Ophthalmology and Neurosurgery, United States Army Hospital, Fort Campbell, Ky.

Many investigators have held that there is a bilateral representation of both maculas. However, most of the evidence at present appears to dispute this concept.

It should be emphasized that many cases of macular sparing are so called because of the examiner's faulty technique. He must be especially careful that the patient does not shift fixation to the moving target as the blind portion of the field is attained. There is a special kind of sparing which Traquair [1] referred to as the "overshot field." In this type the sparing not only is central, but it extends along the whole vertical meridian. This phenomenon is difficult to explain. It is most likely due to faulty fixation. Verhoeff [2] advanced an interesting speculation. He suggested that the optic fixation reflexes are often disturbed in suprageniculate lesions, so that slips in fixation and false sparing are demonstrated. If the optic fixation reflexes are spared, splitting of the macula will be commoner.

Sparing of the macula is commonest in vascular lesions of the occipital cortex and subjacent white matter. This statement is the only assertion which can be made in connection with the localizing value of macular sparing. Supposedly, in anterior lesions (optic tract and anterior radiation) the macula is split, whereas in posterior lesions it is spared. This relation is by no means constant.

Sparing of the macula in lesions of the occipital lobe depends on the nature of the lesion. Occlusive vascular disease usually results in sparing; tumors may spare the macula (although Horrax and Putnam [3] stated that with tumors of the occipital lobe sparing is three times as common as is division of the macula), while injuries rarely spare the macula. Traquair [4] pointed out that traumatic lesions of the occiput spare the macula far less frequently than do vascular lesions. Central vision may be normal, however, since only a portion of the fixation area is involved. Our cases bear out this statement.

Associated Signs and Symptoms.—Another aid in the localizing of lesions of the visual pathways is consideration of the associated signs and symptoms. A discussion of this subject is beyond the scope of the present paper; suffice it to say that hemianopsia due to a radiation defect is usually associated with other symptoms, while occipital lesions are usually "silent" unless there is an obvious scar.

Shape of the Field.—The shape of the field may be important in differentiating lesions of the posterior radiation from lesions of the occipital cortex. Both produce congruous defects. However, the presence of small homonymous hemianoptic scotomas strongly indicates cortical or subcortical damage. The suprachiasmal path is most spread out here, so that a lesion is not uncommonly associated with scotomas. The macular fibers are projected onto the most posterior aspect of the visual cortex. Central and paracentral homonymous scotomas are, then, due to lesions of the posterior cortex. Lesions restricted to this area are rare. Occlusion of small vessels may occur at the pole. This may be seen in the arterial spasm associated with migraine. Tumors rarely arise at the posterior tip. The most usual cause is trauma.

1. Traquair, H.: Clinical Perimetry, St. Louis, C. V. Mosby Company, 1946, p. 250.

2. Verhoeff, F.: A New Answer to the Question of Macular Sparing, Arch. Ophth. **30**:421-425, 1943.

3. Horrax, G., and Putnam, T. J.: Distortions of the Visual Fields in Cases of Brain Tumour, Brain **55**:499-523, 1932.

4. Traquair,[1] p. 276.

Holmes [5] reported a series of such cases. In one of the present cases a paracentral homonymous scotoma was present. Traquair [6] stated that usually the scotoma involves the apex of one quadrant only. This is by no means a rule.

There are finer differences in shape of field which may be helpful in differentiating posterior lesions. A sharp rectilinear delimitation along the vertical and horizontal meridians indicates a lesion of the radiation, while characteristic of cortical defects are an irregular outline and a steep margin. Vascular lesions often result in fields with regular and steep outlines. There has been considerable speculation concerning the clean-cut, straight horizontal margins associated with radiation lesions. Holmes [5] was of the opinion that this indicated that the fibers from the upper and lower retinal quadrants may be separated by an anatomical interval. Roenne [7] believed in the occurrence of such an interval and thought that it was occupied by macular fibers. With all this, it should be borne in mind that although hemorrhage, some injuries, and the early stages of some tumors may implicate one structure without the other, the radiation and the cortex are frequently involved together.

Course of the Lesion.—The course of the lesion may be of aid in diagnosis and localization. Traquair [8] pointed out that when complete blindness is followed by recovery of an entire half-field the occipital cortex is involved, and that sparing of the macula following splitting indicates a vascular obstruction. The early blindness may be due to hemorrhage on one side which creates pressure on the opposite side. It may be noted that in many of the traumatic cases reported below there was initial blindness. The duration of this blindness varies considerably—from minutes to days.

As Walsh [9] stated, a patient so affected may be "blinder than a blind man," since there may be associated visual agnosia or aphasia, loss of stereopsis, and loss of memory for topography. This results from interruption of the vascular supply to the association pathways from the visual cortex.

Relation to Subgeniculate Lesions.—In this discussion we are not concerned with lesions of the tract except as they are related to geniculocalcarine lesions. We have already noted the incongruity of fields with tract lesions. Splitting or sparing of the macula is not a valuable sign. Two other signs are cited in all treatises on this subject. The first, and least important, is the hemianoptic pupillary response of Wernicke. Very few investigators have ever observed this response. The experimental set-up in itself violates many of the principles of optics. Most examiners consider it a useless sign. We concur in this belief. The other sign is optic nerve atrophy. While it is admitted that optic nerve atrophy does occur with subgeniculate lesions, it is of little practical value. It requires several months for the disks to become pale. We are concerned with the diagnosis and localization of the lesion long before this. Of course, atrophy of the optic nerves never develops in supragenicular lesions.

5. Holmes, G.: Disturbances of Vision by Cerebral Lesions, Brit. J. Ophth. **2**:353-384, 1918.

6. Traquair,[1] p. 271.

7. Roenne, H.: Different Types of Defects of the Field of Vision, J. A. M. A. **89**:1860-1865, 1927.

8. Traquair,[1] p. 277.

9. Walsh, F.: Clinical Neuro-Ophthalmology, Baltimore, Williams & Wilkins Company, 1947.

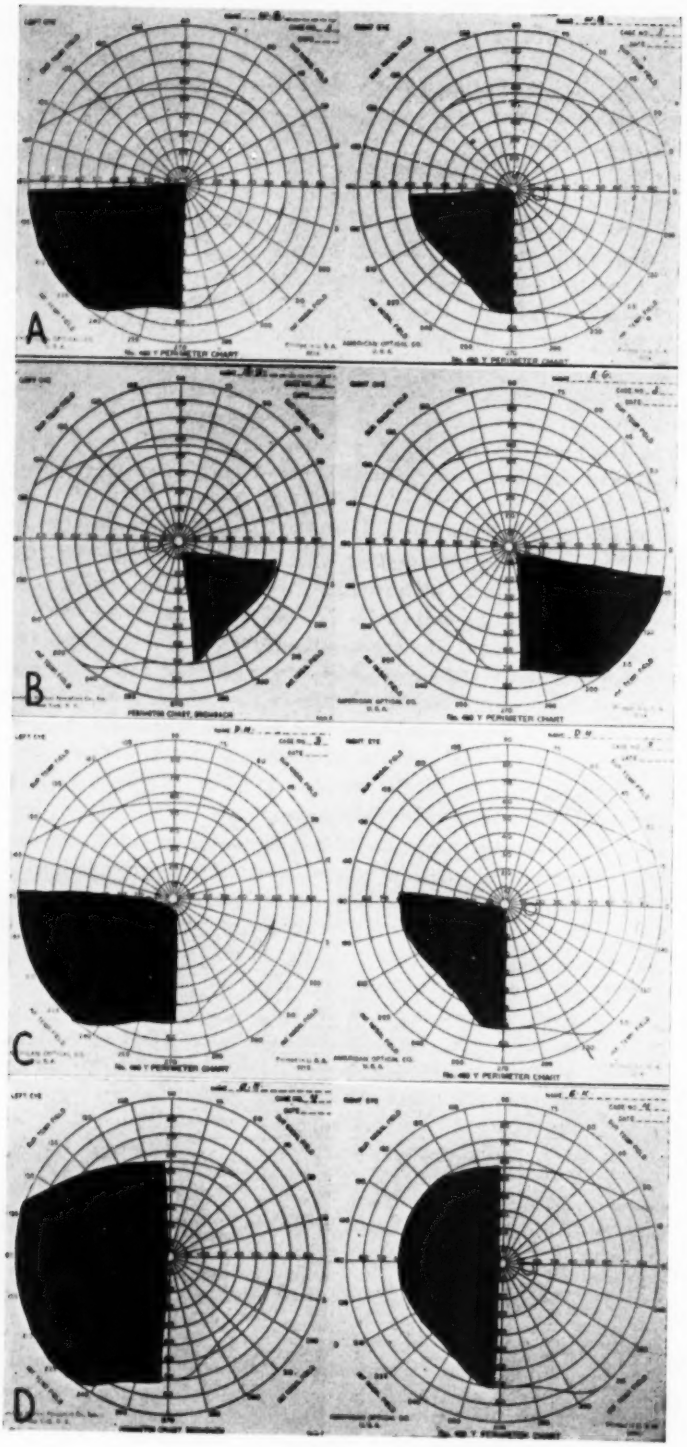

Figure 1

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Bilateral Homonymous Hemianopsia.—Bilateral homonymous hemianopsia is an infrequent occurrence in civilian practice. It may be caused by two separate vascular accidents, one on each side of the brain, ependymitis of both lateral ventricles, tumor arising in the falx between the occipital lobes, or trauma. The vast majority of cases of this defect are seen at military hospitals, and trauma is the etiologic factor.

A transverse bullet wound usually causes a bilateral defect of quadrantic nature. The lower quadrants are involved, since traumatic superior altitudinal defects are caused only by lesions at the base of the posterior part of the brain. The proximity of the major blood sinuses is said to make it impossible for a patient to survive such an injury. In all the cases of bilateral homonymous hemianopsia to be described below the visual field loss was inferior in position.

If the lesion is confined to both occipital poles, bilateral homonymous scotoma will result. Many of the injuries in the present series were occipital, but only one resulted in a hemianoptic scotoma. This is understandable, since missile injuries are most likely to extend anteriorly into the posterior radiations.

All but one of the patients whose cases are described were Korean casualties, and all were treated at the United States Army Hospital, Fort Campbell, Ky., between March, 1951, and February, 1952.

REPORT OF CASES

Homonymous Quadrantanopsias

Case 1.—A white soldier aged 24 was struck by mortar fire on Oct. 21, 1950, in Korea. He sustained a penetrating wound in the right occipital region. There was no loss of consciousness, but the patient remembered visual difficulty immediately following the injury. He was able to distinguish shadows in the upper portion of his field only. He was evacuated to a base hospital, and during the course of evacuation evidence of increased intracranial pressure developed, with left homonymous hemianopsia and moderate blurring of the optic disks. X-rays demonstrated a compound comminuted depressed fracture of the occipital area. Operation, performed on Oct. 28, revealed a 3 mm. hole in the right occipital bone and an underlying dural defect measuring 1 cm. in diameter. Through this dural defect necrotic brain tissue and subcortical clots exuded under pressure. The surrounding dura was extremely tight and showed bluish discoloration. At that time the dura was débrided and the dural opening enlarged. Four comminuted pieces of bone were removed from the shrapnel canal, which ran from the right occiput anteriorly and toward the midline, terminating just before emerging from the cortex at the falx. At a depth of 3 cm. from the cortical surface, a fairly extensive subcortical clot was evacuated by clipping and sharp dissection. Herniated cortex and old necrotic brain tissue were dissected away. A moderately large subdural hematoma was evacuated; the dural graft was placed, and the

---

Explanation of Figure 1

A (Case 1), left lower homonymous quadrantanopsia, congruous in type. Central visual acuity was normal.

B (Case 2), right lower homonymous quadrantanopsia, congruous in type. Central visual acuity was normal. X-rays revealed an old ununited flap fracture in the left posteroparietal region.

C (Case 3), left lower homonymous quadrantanopsia, congruous in type. Central visual acuity was normal. This defect followed a ventricular tap. It was thought that the needle pierced the calcarine fissure.

D (Case 4), left homonymous hemianopsia, congruous in type. Central visual acuity was normal.

wound was closed. The patient made an uneventful recovery and was evacuated to the states. Examination on admission to the United States Army Hospital, Fort Campbell, Ky., revealed left lower homonymous quadrantanopsia, congruous in type.

CASE 2.—A soldier aged 23, in 1945, previous to admission to the service, had a motorcycle accident and received trauma to the left posteroparietal region. Surgical treatment was given at that time, but details are not available. X-ray studies revealed an old ununited flap fracture of the left posteroparietal region. The patient had had persistent headaches ever since the injury. Five months before the present admission, while he was on a field problem just outside Washington, D. C., he was accidentally struck on the head by a tent pin and lost consciousness for a few moments. Since then the headaches had become worse. Examination at the United States Army Hospital, Fort Campbell, Ky., revealed right homonymous inferior quadrantanopsia. He stated that he had had this since the motorcycle accident, in 1945.

CASE 3.—A 19-year-old soldier was evacuated to the states from Europe, arriving at the United States Army Hospital, Fort Campbell, Ky., on July 27, 1951. He complained af headaches, blurring of vision, and a staggering gait. He had been ill for approximately one month and was evacuated with the diagnosis of a possible brain tumor. During his trip from Fort Jay

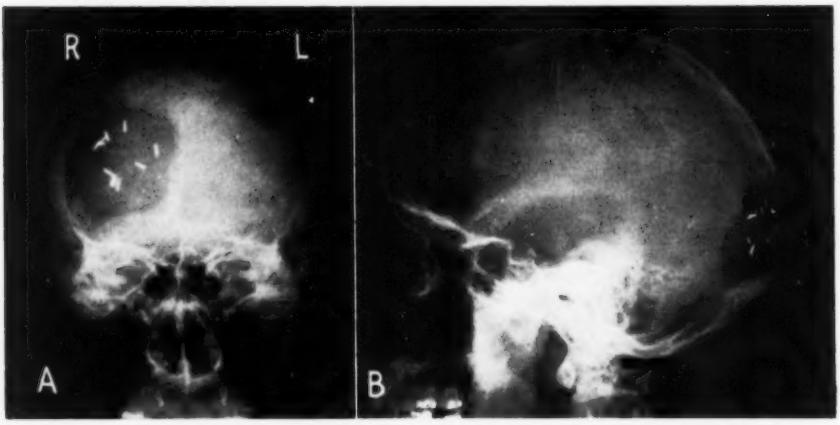

Fig. 2.—*A* (Case 1), anteroposterior roentgenogram of the skull, revealing bone defect on the right side. Wire clips are seen in the defect. A small round burr-hole defect is seen on the left side.

B (Case 1), lateral view revealing the posterior nature of the injury.

to Fort Campbell, the stewardess noted that his breathing was irregular, and he had syncope. His condition was very poor, and the plane was forced to go to a lower altitude because of his dyspnea. Examination at the Army Hospital at Fort Campbell revealed moderate bilateral papilledema, right hemiparesis, and diplopia. A questionable Babinski sign was elicited on the right, and there seemed to be hemihypesthesia on that side. On admission, July 27, 1951, he was extremely nauseated and vomited several times. He seemed to be unimproved, and during the night of July 27 the vomiting became severer and the patient became semicomatose. A tentative diagnosis of neoplasm of the posterior fossa had been made, and on July 28 two burr holes were placed on the occiput and an attempt was made to tap the lateral ventricles. Immediately after the operation the patient claimed that he was blind. The ventricles were not penetrated, but the right-sided weakness improved and the left hemihypesthesia disappeared completely. During the succeeding days the papilledema seemed to subside, and the patient stated he could see shadows. On Aug. 2 left lower homonymous quadrantanopsia was found. This visual field defect showed macular sparing. The patient's general condition gradually cleared, but the quadrantanopsia remained. Vision was 20/20 in each eye. An electroencephalogram revealed cortical disturbances in both occipital areas. A pneumoencephalogram showed

nothing abnormal, and the patient was finally discharged from the Army because of the quadrantanopsia, which, after seven months, was unimproved. His other symptoms disappeared completely. It is believed that the one burr hole that caused the damage was placed almost directly over the midline, and it is possible that the calcarine fissure was pierced by the needle.

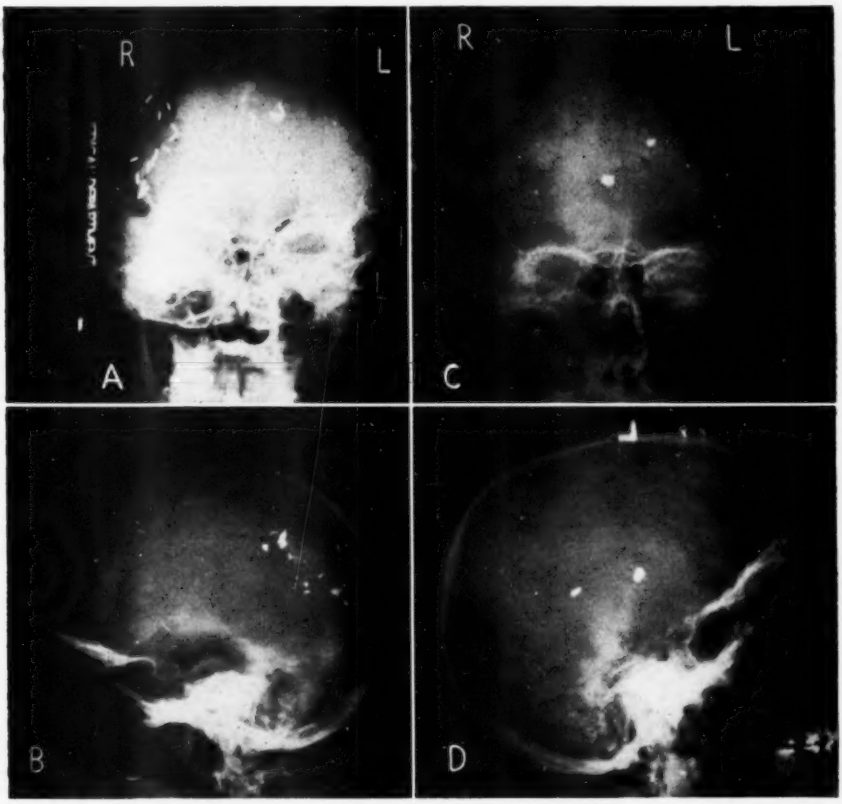

Fig. 3.—*A* (Case 4), anteroposterior roentgenogram revealing tremendous defect on the right side of skull.

B (Case 4), lateral view revealing posterior position of the traumatic injury.

C (Case 5), anteroposterior roentgenogram revealing skull defect on the left side.

D (Case 5), lateral view revealing posterior location of the defect.

Homonymous Hemianopsias

Case 4.—A soldier aged *27* was wounded in action in Korea on July 13, 1951. He was struck by small arms fire in the right parieto-occipital area. A craniotomy was performed by a neurosurgical team the same day, but he remembered nothing from the time he was hit until he awoke after the operation. The right occipital region was explored. Comminuted bone fragments were removed, along with a great deal of liquefied cerebral tissue. A large dural defect was closed with a fascial graft. Postoperative recovery was uneventful, and the patient was evacuated to the states. On admission to the United States Army Hospital, Fort Campbell, Ky., the only findings were a bony defect in the right posterior parieto-occipital region and left homonymous hemianopsia with macular sparing. This field defect persisted without change, and the patient was finally discharged from the service.

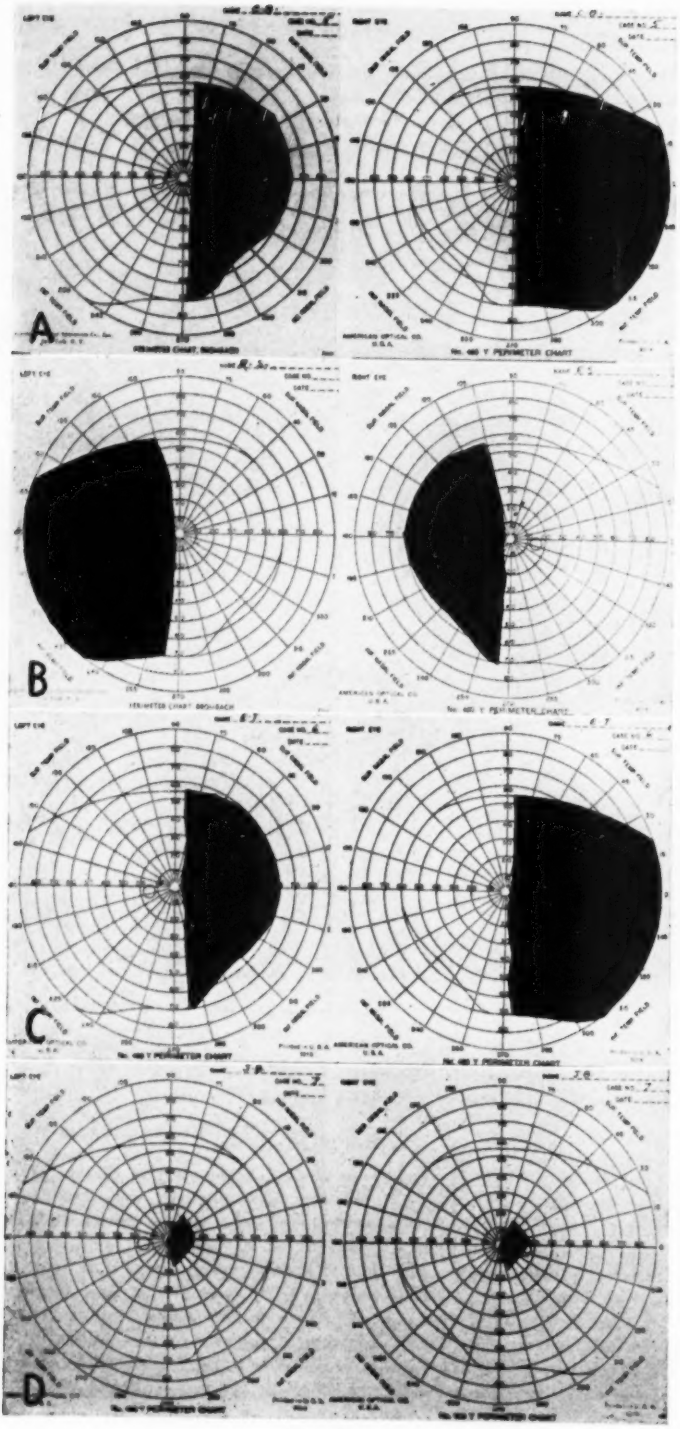

Figure 4

(See legends on opposite page)

CASE 5.—A soldier aged 20 was wounded in action in Korea on Sept. 27, 1951. He did not remember being wounded but was told afterward that he had been hit in the back of the head by enemy shrapnel. The day following his injury he was seen by a provisional neurosurgical team. A ragged laceration 4 cm. in diameter was noted in the left posterior parietal region. He was conscious but confused and irrational, and a right homonymous hemianopsia was present. Roentgenograms of the skull revealed a compound comminuted depressed fracture in the left occipital region of the skull, with bone fragments indriven. A craniectomy was performed. At operation, a punched-out area of bone, 1 cm. square, was revealed in the left occipital area. Craniectomy was carried out medially, and all necrotic cerebral tissue and blood clot, along with bone fragments, were removed. The dural defect was closed with a temporal fascial graft, and the patient made an uneventful recovery, with no complications. He was finally evacuated to the states. On admission to the United States Army Hospital, Fort Campbell, Ky., it was found that his concentration was poor and he had forgotten almost all his experiences in Korea. Examination revealed a vertical scar, 4 in. (10 cm.) long, over the left occipital region, beneath which there was a 5 cm. skull defect. The rest of the examination was noncontributory except for slight confusion and difficulty in calculating and concentrating. Ophthalmological examination revealed right homonymous hemianopsia. An electroencephalogram, on Nov. 15, 1951, revealed slow activity, limited to the left occipital area. After convalescence, the patient was discharged from the Army to a Veterans Hospital for further rehabilitation.

CASE 6.—A white man aged 27 was admitted to the Tokyo Army Hospital on Dec. 16, 1951, in transfer from the neurosurgical detachment at the 121st Evacuation Hospital in Korea. The history revealed that he was wounded in action in Korea on or about Oct. 6, 1951, sustaining a perforating wound of the skull, with the point of entrance in the right temporal region and the exit in the midparietal area at the midline. He was admitted to the neurosurgical detachment on Oct. 6, at which time he was found to be disoriented but conscious and able to speak. There was a large cerebral herniation in the right temporoparietal area. The right eye was deviated laterally, and the right pupil was dilated and fixed. There was complete flaccid left hemiplegia, which included the face. On Oct. 7, a right frontotemporoparietal craniectomy was done. At the time of operation the superior sagittal sinus was found to have been thrombosed and lacerated. There was extensive destruction of the cerebrum in the right frontoparietal area, involving the greater part of the motor strip. Many bone fragments and much devitalized cerebral tissue were removed. Dural closure was achieved with a fascia-lata graft. Postoperative rhinorrhea developed on the left side; this subsided spontaneously. The patient's postoperative course was rather stormy. X-rays revealed the presence of retained bone fragments, and on Oct. 20 a second craniectomy was done, with the removal of three bone fragments. The postoperative course was again rather stormy, but the patient made progressive improvement, and on Nov. 26 a second operation was done, with the evacuation of a large subcortical hematoma. The patient made a slow but progressive recovery. A large accumulation of fluid beneath the skin in the right frontoparietal area was treated with frequent flap and spinal punctures. The scalp flap was noted to bulge. However, the condition in this area became stabilized, and, according to available records, no flap or spinal puncture was done in the two weeks prior to his transfer to Tokyo Army Hospital. On his admission to this hospital the patient was conscious, fairly alert, but not well oriented. A healed incision in the right fronto-

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EXPLANATION OF FIGURE 4

A (Case 5), right homonymous hemianopsia, congruous in type. Central visual acuity was normal.

B (Case 6), left homonymous hemianopsia, congruous in type. Central visual acuity was normal. This patient sustained a perforating wound of the skull with point of entrance in the right temporal region and exit in the midparietal area at the midline.

C (Case 7), right homonymous hemianopsia, congruous in type. Central visual acuity was normal. The patient had received a penetrating wound of the brain, left parietal area, transventricular.

D (Case 8), right homonymous paracentral scotoma. Central visual acuity was normal.

temporal area extended just to the left of the midline in the midparietal area. The scalp flap bulged approximately 1.5 cm. above the surface of the adjacent scalp. One gained the impression of palpable underlying fluid.

The pupils were equal and reacted to light and in accommodation. There were complete left homonymous hemianopsia and left facial paresis of central type. The patient was right-handed. He had flaccid left hemiplegia, including the face. The deep reflexes were hyperactive on the left, and Babinski sign was elicited. There appeared to be left hemihypalgesia and cortical sensory diminution. Meningeal signs were absent. Vital signs were within normal limits. The rest of the physical examination revealed an essentially normal condition. The flap area was tapped, but only a few cubic centimeters could be obtained. One gained the clinical impression of bulging of the flap due to a large porencephalic cyst, with fluid in the subdural area.

The patient was then placed in the lateral position. A spinal puncture was done, the initial pressure being 190 mm. of cerebrospinal fluid. The fluid was slightly xanthochromic but clear; 80 cc. of fluid was removed. The scalp flap became concave and pulsated well. The final pressure was 40 mm. of fluid. Culture of the subgaleal fluid was reported to yield a hemolytic Staphylococcus. This organism was found to be sensitive to all antibiotics but was thought to represent a contaminant, as there had been no evidence of infection. Spinal fluid withdrawn at the time of the puncture remained sterile and contained 20 cells per cubic millimeter. The total protein content was 190 mg. per 100 cc. X-rays taken of the skull on admission revealed two tiny bone fragments above the lower margin of the operative defect on the right side. At the time of this report the patient had been up daily in a chair. His cerebral defect remained concave. The hemoglobin was 16.3 gm. per 100 cc. Urinalysis showed occasional white cells per high-power field. There were no parasites or ova in the stool. The patient had been kept on antibiotic therapy for the week prior to admission because of the positive culture of the subgaleal fluid. He was fairly alert and did not complain of headaches. The left hemiplegia persisted.

Case 7.—A white man aged 22 was admitted to Tokyo Army Hospital Jan. 26, 1952, in transfer from a neurosurgical detachment in Korea. The history revealed that he was wounded in action in Korea by enemy mortar fire on Jan. 7, 1952, and was admitted to the 2d Neurosurgical Detachment 19 hours after injury. He sustained a penetrating wound of the brain, left parietal area, which was transventricular. On admission to the neurosurgical detachment he was stuporous but responded by opening his eyes. He was aphasic. From a 2-cm. penetrating wound in the left parietal area cerebral tissue exuded. The face was symmetrical but did not respond to painful stimulation on the right. The pupils were dilated and equal and reacted to light. There was right hemiplegia of hypotonic nature. The deep reflexes were hyperactive on the right. The abdominal reflexes were absent bilaterally. The Babinski response was elicited on the right. Nuchal rigidity was present. X-rays of the skull revealed a depressed fracture, left parietal area, with deeply indriven bone fragment. On Jan. 8, 1952, a left parietal craniectomy was done and a right occipital burr hole placed. The missile tract was débrided of bone fragments and necrotic cerebral tissue. Three separate tracts were found, all of which contained bone fragments; these were removed. A right occipital burr hole was placed to rule out a subdural hematoma. None was found. Repeated postoperative lumbar punctures were made to insure sealing of the dural graft. Postoperative roentgenograms of the skull revealed the presence of a retained bone fragment. This was removed on Jan. 11, 1952. A subgaleal hematoma developed, which was removed on Jan. 13. On Jan. 24 a subgaleal collection of ventricular fluid developed. The patient was treated by flap and spinal taps after that. He was transferred to Tokyo Army Hospital on Jan. 26, 1952, with the recommendation that flap and spinal taps be continued in an effort to seal the dural graft. Examination on his admission to this hospital revealed an inverted U-flap in the left parietal area which was bulging slightly. The fluid could be palpated beneath its surface. The patient was alert and rational, but aphasic; he understood spoken words and was able to read and follow simple commands.

The pupils were equal and reacted to light and in accommodation. Funduscopic examination revealed clear-cut disk margins with an arterioventricular ratio of 2:3 on the left and 2:4 on the right. The extraocular muscles were intact, and right seventh nerve paralysis of central type was noted. The tongue protruded in the midline.

The patient was right-handed. Examination showed right hemiplegia, diminished deep reflexes on the right, and absence of the Babinski and Hoffmann signs. The patient responded to pain; there was questionable cortical sensory loss on the right. Meningeal signs were not elicited. The

chest, abdomen, and extremities were normal. A flap puncture was done on admission, at which time 40 cc. of watery, clear, slightly xanthochromic fluid was removed. After the flap puncture, a spinal tap was done. The initial pressure was 120 mm. The fluid was clear and slightly xanthochromic. Postoperative roentgenograms of the skull revealed a questionable superficial bone fragment in the left parietal area and a foreign body in the right occipital region, near the surface. A left lateral stereoscopic roentgenogram showed a small bone fragment lying adjacent to the inner table of the skull. Examination of the spinal fluid on admission showed 11 cells and a total protein content of 53 mg. per 100 cc., and culture of subgaleal fluid taken on Jan. 26, 1952 was sterile. There was 78 cells per cubic millimeter in the subgaleal fluid. The patient was treated by combined flap and spinal tap and pressure air dressings for several days. Culture of the subgaleal fluid taken on Jan. 28 yielded a Staphylococcus and a Staphylococcus was also cultured from the spinal fluid on that date. The laboratory workers thought that this growth represented a contaminant. The spinal fluid on Feb. 1 showed 18 cells, and the subgaleal fluid 26 cells, per cubic millimeter. Cultures were sterile, and repeated cultures of the spinal and subgaleal fluid, taken on Jan. 31, Feb. 1, and Feb. 3, were sterile. The spinal

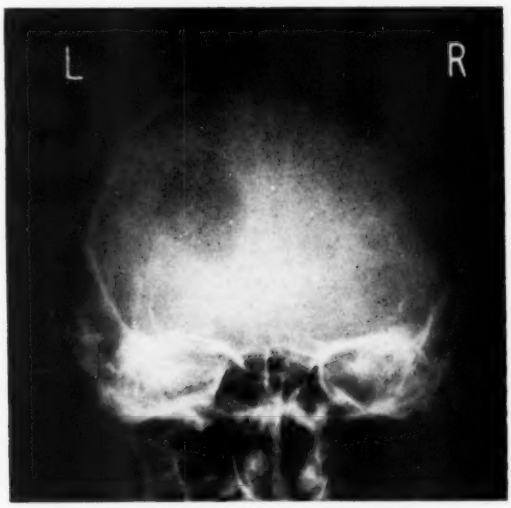

Fig. 5 (Case 8).—Anteroposterior roentgenogram revealing defect on left side of skull. Note that the film has been turned around. The original x-ray, taken in Korea, revealed a depressed fracture of the skull in the left occipital region.

fluid was last cultured on Jan. 4, 1952, at which time it was sterile. There were 90 cells per cubic millimeter and a protein content of 65 mg. per 100 cc. The flap has remained flat since. The patient was kept on a regimen of large doses of chloramphenicol (Chloromycetin) until Feb. 13, 1952. There was no evidence of intracerebral infection, meningitis, or increased intracranial pressure, and use of antibiotics was discontinued on Feb. 13, 1952, so that he could be observed for a period before evacuation. There has been a return of speech, and, although moderate motor aphasia is still present, there has been striking improvement in the speech since admission. The right leg has shown some improvement in motor power, but there is still marked paresis of the right arm. Visual fields reveal right homonymous hemianopsia.

Paracentral Homonymous Scotoma

Case 8.—On Jan. 1, 1951, while a prisoner of the Chinese Communists, a 19-year-old soldier was struck in the back of the head by shrapnel, apparently from artillery fire. He was unconscious for a short time and on recovery found that he was completely blind. He was released and reached an American air station on the following day. Twenty-four hours after injury his

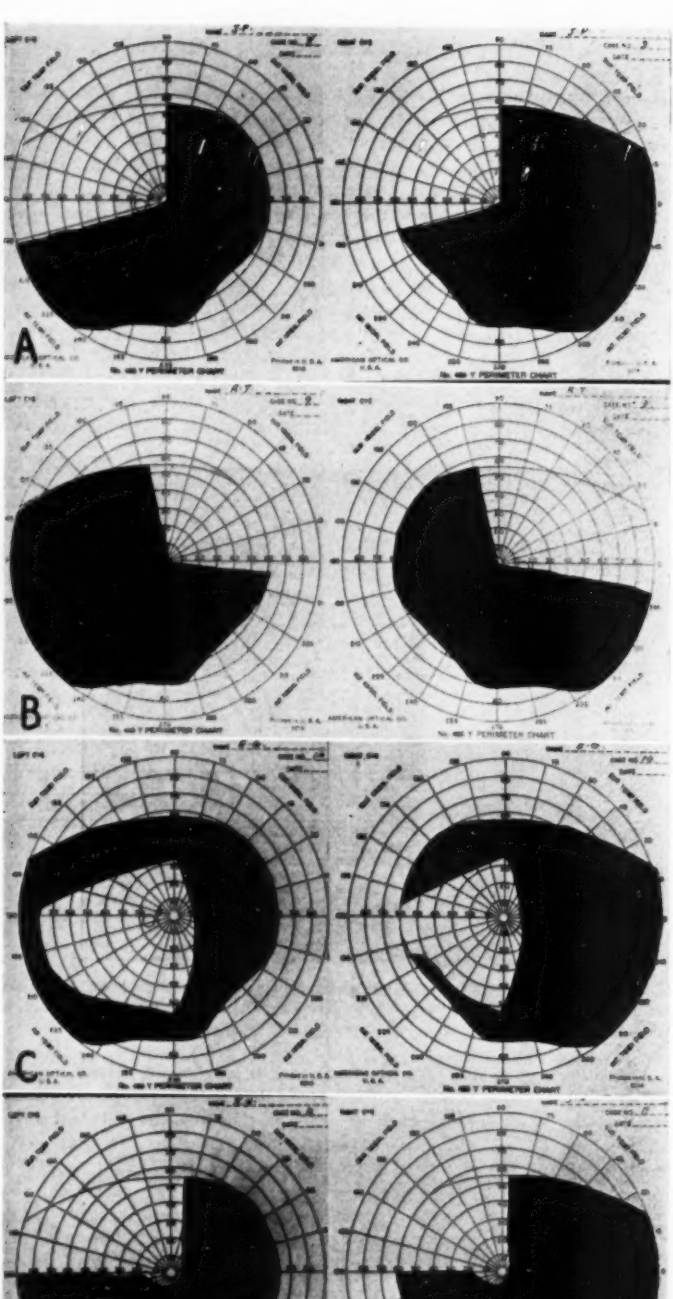

Figure 6

(See legends on opposite page)

vision began to return. He was taken to a station hospital, where, on Jan. 6, 1951, x-rays revealed a depressed skull fracture in the left occipital region, the area of depression measuring 2.5 by 4 cm. and lying just to the left of the midline, and just above the superior nuchal line. A craniectomy was done. Bone fragments and a subdural clot were removed, and the defect in the dura was repaired. Recovery was uneventful, without complications. Vision cleared completely except for right congruous hemianopsia, scotomatous in type, which has remained stationary since his admission to the United States Army Hospital, Fort Campbell, Ky. The operative report from Korea revealed that during repairing of the dural defect a bone fragment had penetrated the dura. Extensive operative details are not available because of the situation involving his injury.

Bilateral Homonymous Hemianopsias

Case 9.—A 29-year-old soldier was wounded in action in Korea, April 10, 1951, when he was struck by shrapnel from an enemy grenade. At operation, on the day following his injury, it was noted that he had an avulsed, gaping wound in the midocciput, which was grossly contaminated, with necrotic cerebral tissue presenting through the wound. Multiple comminuted bone fragments had been driven into the brain. Craniectomy, carried out in the left occipital region across the midline, showed that one of the bony fragments had punctured the superior sagittal sinus. This injury was repaired with muscle, and, after débridement of the left occipital lobe, a considerable quantity of brain and necrotic tissue with bone fragments was sucked out along the medial surface of the lobe. Examination of the right occipital lobe revealed multiple contusion hemorrhages. No brain tissue was removed, and the dura was closed. The patient made an uneventful recovery and was evacuated to the states for further therapy and rehabilitation. Examination at the United States Army Hospital, Fort Campbell, Ky., revealed that immediately after injury the patient was totally blind and that the first thing to return was light perception, about one week after operation. About one week later he was able to distinguish forms. He had no headaches or seizures, and the only abnormal finding was right homonymous hemianopsia and left inferior quadrantanopsia. Further convalescent care produced no change in the patient's ophthalmological condition, and he was finally discharged from the hospital and returned to limited duty.

Case 10.—A soldier aged 23 was wounded in action in Korea on June 3, 1951, when a hand grenade exploded near him. The concussion of the grenade threw him to the ground, and he noted immediately that he was blind in both eyes. He did not lose consciousness at any time and was fully aware of what was going on around him. He was evacuated to Taegue, and the following day a bioccipital craniectomy was performed for débridement of the penetrating wound in the right occipital area. It was noted during the craniectomy that multiple indriven bone fragments were scattered intracerebrally along the side of the falx on the right. Multiple small bone fragments adhered to the edge of the dural tear in the occipital region and along the edges of the sinus. When the bone fragments were removed, the sinus was found to be completely unroofed for approximately 0.5 cm., and each end was almost totally thrombosed. Several sutures were placed in the wall of the sinus, and a piece of fibrin film was tied over it. There was a large intracerebral tract of the missile with about 10 bone fragments in it. These were removed; the necrotic brain was sucked out, and bleeding points were controlled with cautery. A dural graft was applied, and the patient made an uneventful recovery. On the second post-

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Explanation of Figure 6

A (Case 9), right homonymous hemianopsia with left lower quadrantanopsia, congruous in type. Central visual acuity was normal.

B (Case 10), left homonymous hemianopsia with right lower homonymous quadrantanopsia, congruous in type. Central visual acuity was normal.

C (Case 11), right homonymous hemianopsia with involvement also of the left visual fields. The trauma resulted in bone fragments driven into the left parietal area. The defect is congruous in type; central visual acuity was normal.

D (Case 12), right homonymous hemianopsia with left lower homonymous quadrantanopsia, congruous in type. Central visual acuity was normal.

Fig. 7 (Case 9).—Roentgenogram of the skull revealing a large defect on each side of the midline, more extensive on the left.

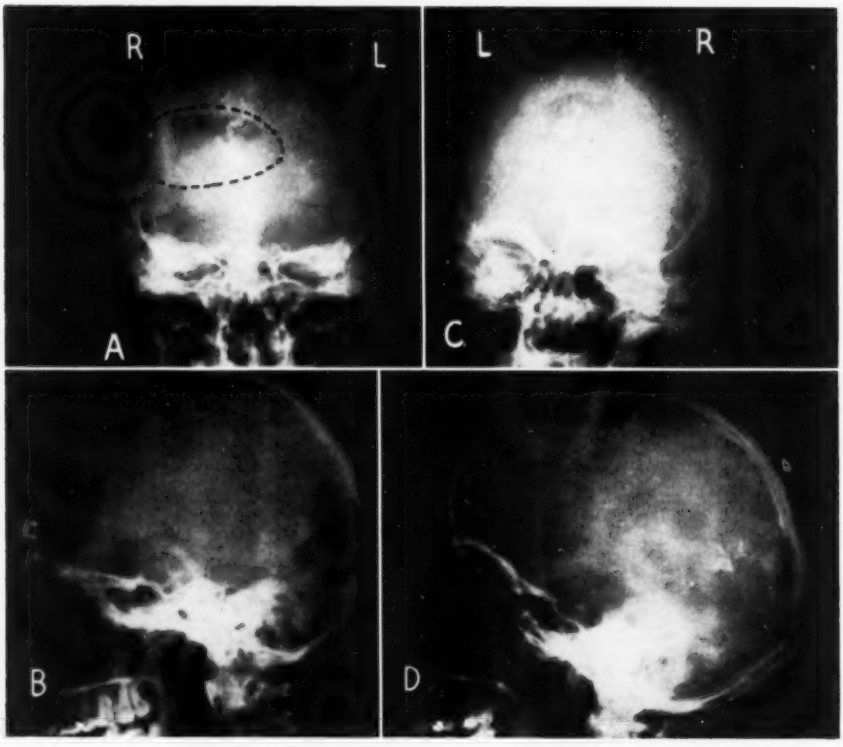

Fig. 8.—*A* and *B* (Case 10), roentgenograms of the skull revealing a defect on the right side, extending slightly across the midline.

C and *D* (Case 12), roentgenograms of the skull which are turned around, revealing a defect on the right side posteriorly.

operative day he was able to make out objects to his left. He was transferred to Tokyo Army Hospital for further convalescent care. He was then returned to the States, arriving at the United States Army Hospital, Fort Campbell, Ky., July 4, 1951. Examination on admission showed a linear transverse cranial defect across the occipital region, approximately 2 by 6 cm. Examination of the visual fields on July 7, 1951, revealed that only one-fourth of his visual fields, the upper right quadrant, was left in each eye. This indicated that he had severe damage to the right occipital lobe and to the upper fibers in the left occipital lobe. Other than the field defect and the findings noted above, no abnormalities have been noted. The patient underwent tantalum cranioplasty, for cosmetic purposes only, and was discharged from the Army.

CASE 11.—A soldier aged 19 was wounded by enemy machine-gun fire in Korea on March 14, 1951. Two machine-gun bullets struck the top of his head, and the next thing he remembered he was in a helicopter. There he lost consciousness again, regaining it several days later on the U. S. S. *Repose Hospital Ship*, at Pusan. At the time of injury bone fragments were driven into the left parietal region, and an operation performed on the hospital ship revealed severe laceration, and contusion of the brain, notably in the left parietal and occipital lobes. A craniectomy, with an opening about the size of a silver dollar, was performed in the posteroparietal region, and the bone fragments were removed. A small portion of brain was sucked out. The dural defect was closed, and the patient made an uneventful recovery. On admission to the United States Army Hospital, Fort Campbell, Ky., the patient had moderate aphasia and mild right hemiparesis. Ophthalmological examination revealed an extensive bilateral visual field defect. After several months at this hospital, reexamination from time to time showed no change in his neurological or ophthalmological condition. He was subsequently discharged to a Veterans Hospital for further rehabilitation.

CASE 12.—A soldier aged 20 was wounded in action in Korea on May 23, 1951. Immediately after his injury he was disoriented and confused. There was a gaping wound, 3 cm. in width, in the left occipital region with visible cerebral tissue in the depth of the wound. His condition was complicated by a penetrating wound of the left side of the thorax, with almost complete hemothorax on admission. Craniectomy was performed on the left occipital area one week after injury. At craniectomy, when the bony opening was enlarged to permit débridement of the brain, the tract of the missile was explored and several bony fragments were removed. There was a gush of necrotic brain tissue and pus, amounting to 10 or 15 cc. Several additional bony fragments were felt deep in the tract of the missile and removed. Palpation revealed a large area of cerebral softening in the left occipital lobe. This tissue was sucked out and resection carried back to firm brain tissue. It did not extend to the ventricle. The postoperative course was rather stormy. Meningitis developed and was treated with penicillin, streptomycin, and sulfadiazine. The infection finally cleared up. He was evacuated to the states for further rehabilitation. Examination at United States Army Hospital, Fort Campbell, Ky., revealed that, except for a draining wound in the left occipital region, which required secondary closure, and a severe visual field defect, the patient was in relatively good physical condition. After a period of convalescence, he was discharged to a Veterans Hospital for further care.

SUMMARY

This report is concerned with visual field defects due to trauma. They are typical of those seen in the current conflict.

The lesions discussed are restricted to those involving the geniculocalcarine pathway. The following factors are most important in the localization of the lesion:

Congruity.—The most important single point of differentiation between homonymous hemianopsias due to anterior lesions (optic tracts and anterior radiations) and those due to posterior lesions (posterior radiations and occipital cortex) is the remarkable congruity of the visual field patterns of the two eyes with the latter type. Congruity implies similarity of all portions of the fields except the temporal crescents.

Sparing of the Macula.—This sign is of considerably less value. Sparing occurs typically in posterior lesions. It is commonest in vascular lesions, less common in tumors, and least common in trauma. In cases of trauma the macula is usually split, central visual acuity remaining good.

Associated Signs and Symptoms.—This factor is important in localization. Occipital lesions are usually "silent," while radiation lesions are almost always associated with other signs and symptoms.

Shape of the Field.—The presence of small homonymous hemianopsic scotomas strongly indicates cortical or subcortical damage. The suprachiasmal path is most spread out here, so that scotomas are common. Trauma is the most frequent cause. A sharp rectilinear delimitation along the vertical and horizontal meridians indicates a radiation lesion, while an irregular outline and a steep margin are characteristic of cortical defects. Vascular lesions often result in fields with regular and steep outlines.

Course of the Lesion.—When complete blindness is followed by recovery of an entire half-field, the occipital cortex is usually involved. When sparing of the macula follows splitting, a vascular obstruction is indicated. The early blindness may be due to a hemorrhage on one side which creates pressure on the opposite side. The length of this initial blindness varies considerably—from minutes to days.

Relation to Subgeniculate Lesions.—Subgeniculate lesions usually show incongruity. Splitting of the macula is not a reliable sign. The hemianoptic pupillary response of Wernicke is difficult to elicit. Optic nerve atrophy occurs, but the process takes considerable time to reach the optic disk.

Bilateral Homonymous Hemianopsia.—This defect may be caused by two separate vascular accidents, one on each side of the brain; ependymitis of both lateral ventricles; tumor arising in the falx between the occipital lobes, and trauma. Transverse bullet wounds will usually cause a bilateral defect of a quadrantic nature. The lower quadrants are involved, since superior altitudinal defects of traumatic origin are caused only by lesions at the base of the posterior part of the brain. The proximity of the major blood sinuses makes it virtually impossible for a patient to survive such an injury.

Cases of the following categories are described: (1) homonymous quadrantanopsia, 3 cases; (2) homonymous hemianopsias, 4 cases; (3) homonymous paracentral scotoma, 1 case, and (4) bilateral homonymous hemianopsia, 4 cases.

BILATERAL ENDOPHTHALMITIS PHACOANAPHYLACTICA

Pathologic Study of the Lesion in the Eye First Involved and, in One Instance, the Secondarily Implicated, or "Sympathizing," Eye

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FOR MANY years I have noted that enucleated eyes referred for histologic study and found to show the lesion called by Verhoeff and Lemoine [1] endophthalmitis phacoanaphylactica have occasionally been submitted under the clinical diagnosis or suspicion of sympathetic ophthalmia. In some such cases inflammation in the second eye has been unusually severe and persistent. This experience led to the conviction that endophthalmitis of this type might be bilateral, and the concept became well known among practitioners and students at the Brooklyn Eye and Ear Hospital. It was not until 1951, however, that an opportunity presented to establish the diagnosis histologically by examination of an enucleated second, or "sympathizing," eye in such a case. In all our previous cases the nature of the lesion in the second eye, whether lens-induced endophthalmitis, true sympathetic ophthalmia, or some other type of disease, remained without histologic verification. The "proved case" and selected examples of "presumptive" cases of bilateral endophthalmitis phacoanaphylactica are presented below, with special consideration of the varied histologic character of the condition.

In 1942, Courtney [2] published a report of seven cases of bilateral inflammation following in each instance extracapsular operation for a senile cataract. The second eye, in each case, had not been traumatized. Cutaneous sensitivity to lens antigen was demonstrated in all seven cases, and in one of the cases examination of the enucleated second eye showed a picture suggestive of endophthalmitis phacoanaphylactica. Courtney was evidently the first to publish the concept that a bilateral form of the disease exists.

Without knowledge of Courtney's paper, I [3] reported, in 1947, the finding of endophthalmitis phacoanaphylactica in the "exciting" eye in a case of bilateral uveitis that followed extracapsular extraction and pointed out that this case might be one of bilateral lens-induced inflammation, or that true sympathetic ophthalmia had supervened.

This paper was awarded the William Browning Prize for 1952.

From the Departments of Pathology of the Brooklyn Hospital and the Brooklyn Eye and Ear Hospital.

1. Verhoeff, F. H., and Lemoine, A. N.: Endophthalmitis Phacoanaphylactica, Tr. Internat. Cong. Ophth. **1**:234, 1922.

2. Courtney, R. H.: Endophthalmitis with Secondary Glaucoma Accompanying Absorption of the Crystaline Lens, Tr. Am. Ophth. Soc. **40**:355, 1942.

3. deVeer, J. A.: Sympathetic Ophthalmia vs. Phacoanaphylaxis, Society Proceedings, Am. J. Ophth. **31**:1004, 1948.

In April, 1952, Haik, Waugh, and Lyda [4] described a case in which the clinical interpretation was sympathetic ophthalmia, whereas examination of the enucleated "second" eye had shown a picture similar to that in Courtney's [2] case. The diagnosis of bilateral endophthalmitis phacoanaphylactica was made. Unfortunately, the lens was lost during handling of the tissue, and the illustrations published do not, therefore, show a textbook type of lesion, though the interpretation was undoubtedly correct. Haik and his co-authors reviewed reported cases of "sympathetic ophthalmia" in which the second eye had been examined and concluded that "clinical and histologic evidence in these cases suggests that a bilateral form of endophthalmitis has apparently been confused clinically with sympathetic ophthalmia." They also pointed out that the composite histologic picture in the reports reviewed was strikingly like that in their own case, implying that there may have been confusion also in the histologic interpretation of cases reported as instances of sympathetic ophthalmia. These observations spurred me to attempt to fortify the concept that endophthalmitis phacoanaphylactica may occur bilaterally and that the lesion in the second (nontraumatized) eye, in its typical form, is histologically identical with the lesion found in the unilateral disease. That is the purpose of the present communication. It is offered as a corollary to a study of the lesion of sympathetic ophthalmia in a sympathizing eye,[5] a histologic syndrome in sharp contrast to the one here depicted.[6]

ETIOLOGY

No review of the much-debated question of the etiology of the endophthalmitis under discussion will be attempted, but it may be pointed out that sensitivity to lens protein was put forth as the important causative factor by Verhoeff and Lemoine [1] in 1922, when the name endophthalmitis phacoanaphylactica was proposed. Infection and a toxic influence of lens substance have both been emphasized by those who have opposed the allergic theory. Whether or not sensitivity to lens protein be considered the principal factor involved, it is generally agreed that there is an allergic aspect to the condition, as evidenced by the characteristics of the lesion and by sensitivity to lens antigen demonstrable by skin testing. The more noncommittal term "lens reaction," or "lens-induced endophthalmitis," is preferred by many because of the possible influence of etiologic factors other than allergic ones.

PATHOGENESIS

The mechanism of development of the inflammation in the bilateral form of the disease is probably essentially the same as in cases of the unilateral form except for the likelihood that initial sensitization is the result of liberation of lens substance

4. Haik, G. M.; Waugh, R. L., Jr., and Lyda, W.: Sympathetic Ophthalmia: Similarity to Bilateral Endophthalmitis Phacoanaphylactica; New Therapeutic Methods, A. M. A. Arch. Ophth. **47**:437, 1952.

5. deVeer, J. A.: Sympathizing Eye in Sympathetic Ophthalmia, A. M. A. Arch. Ophth. **48**:723, 1952.

6. The papers of Courtney and of Haik and others and this offering all distinguish two distinct pathologic entities which may be clinically confused. There is, in my opinion, a third form of disease, in which a "lens-induced," or phacoanaphylactic, endophthalmitis is accompanied with or followed by true sympathetic ophthalmia (deVeer, J. A.: Endophthalmitis Phacoanaphylactica and Its Relation to Sympathetic Ophthalmia, Arch. Ophth. **23**:237, 1940). A discussion of this combination of lesions and their possible etiologic relationships has been omitted from the body of this paper to avoid confusion.

from the eye first traumatized by operation or accidentally. Sensitization to lens protein without perforation of the capsule may be a possibility, but that question has not been satisfactorily settled and will not be discussed except to point out that rupture of the capsule is usually demonstrated or assumed in unilateral cases and was believed to have occurred in the "second" eye, presumably spontaneously, in Courtney's histologically examined specimen, in the case reported by Haik and his associates, and in the histologically verified case reported below (Case 3). This would suggest that bilaterality of lens-induced endophthalmitis is much the same as bilateral occurrence of tuberculosis or other diseases, without the mysterious element that has proved so baffling in sympathetic ophthalmia, namely, the manner of "extension" from one eye to the other. When both eyes have been subjected to operative procedures, it may not be possible to learn which eye initially gave rise to sensitivity to lens protein, and, therefore, which was the "exciting" eye. It might be more correct to refer to the eye first to show inflammation as the "first" eye and the one subsequently involved as the "second" eye.

It has been noted that in this condition the involvement of the second eye has usually been a delayed reaction, appearing at a time when the first eye was no longer inflamed. On the other hand, in sympathetic ophthalmia there is almost always simultaneous inflammation in the two eyes.

THE LESION OF ENDOPHTHALMITIS PHACOANAPHYLACTICA

In the unilateral, and also, as will be shown, in the bilateral form, the disease is essentially an inflammation centering on a site of disintegration of lens substance where a break in the capsule has permitted escape of lens material, or entrance of inflammatory elements, or both. At this center acute inflammatory exudate collects in small or larger amounts and tends to become walled off by a zone of granulomatous inflammation. The vascular neighboring tissues, notably the iris and the ciliary body, are the major source of the leucocytes and other inflammatory elements, and they themselves are involved in an infiltration with plasma cells and, often, lymphocytes, in nodular aggregations. A more remote and usually mild, or even insignificant, infiltration, chiefly with lymphocytes, occurs in the limbic episcleral tissue, the choroid, the retina, and the optic nerve head.

Viewed for purposes of analysis in this oversimplified way, the picture resembles that of a well-localized abscess, with distinct zones of reaction, and might profitably be represented in the schematic way that has long been employed in the description of the abscess. The center of the disturbance contains not bacteria but lens matter, which can be identified as lens fibers, droplets of Morgagni, or fragments of nucleus. It probably contains also liquefied lens substance, which, aside from being eosinophilic, is not distinguishable from the fluid component of exudate with which it is mixed. If the lesion is considered an allergic phenomenon, or one in which there is a significant allergic factor, the lens material may be regarded as a visible, recognizable etiologic factor (antigen), comparable to the recognizable ocular melanin in the lesions of sympathetic ophthalmia or in a positive Friedenwald skin reaction. Among and about these lens derivatives, acute inflammatory exudate with a predominance of neutrophilic polymorphonuclear leucocytes collects, and the component cells undergo more or less disintegration. This site is represented in the accompanying diagram as the center of a series of circles (Fig. 1).

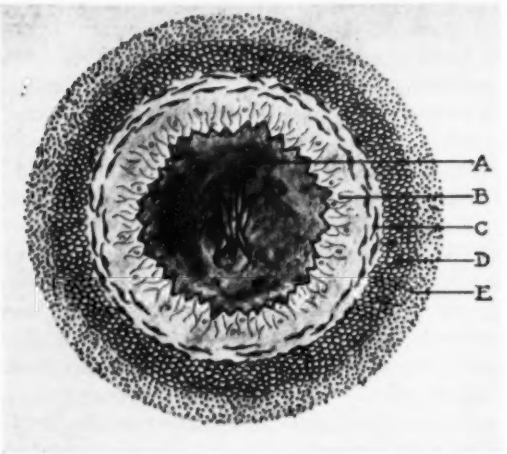

Fig. 1.—Diagrammatic representation of the lesion of endophthalmitis phacoanaphylactica. *A*, indicates purulent center containing identifiable lens derivatives; *B*, bordering fringe of epithelioid structures; *C*, fibrovascular zone; *D*, zone of plasmocytic infiltration; *E*, zone of lymphocytic infiltration.

Fig. 2.—(*a*) In a pocket partially enclosed by lens capsule, remnants of lens fibers are surrounded by acute exudate. There is also exudate in the anterior chamber into which lens substance has escaped. An encapsulating layer of macrophages and epithelioid tissue is tending to seal off the pocket. (*b*) The pupillary portion of the iris has served as a major source of inflammatory exudate. It has become adherent to the lens mass by the production of a layer of granulation tissue with a fringe-like epithelioid border. The eye was struck by a piece of wood seven weeks prior to enucleation.

When one keeps in mind (*a*) the avascular state of much of the ocular mass, (*b*) the presence of glass membranes, which limit or prevent escape of exudates into the chambers from extensive vascular areas, and (*c*) the influence of adhesions, which tend to block off both escape of antigen and movement of exudates, it is not surprising that the "center" of the lesion is not geometrically central and that the "zones" of reaction are not always encircling, except in a diagrammatic or theo-

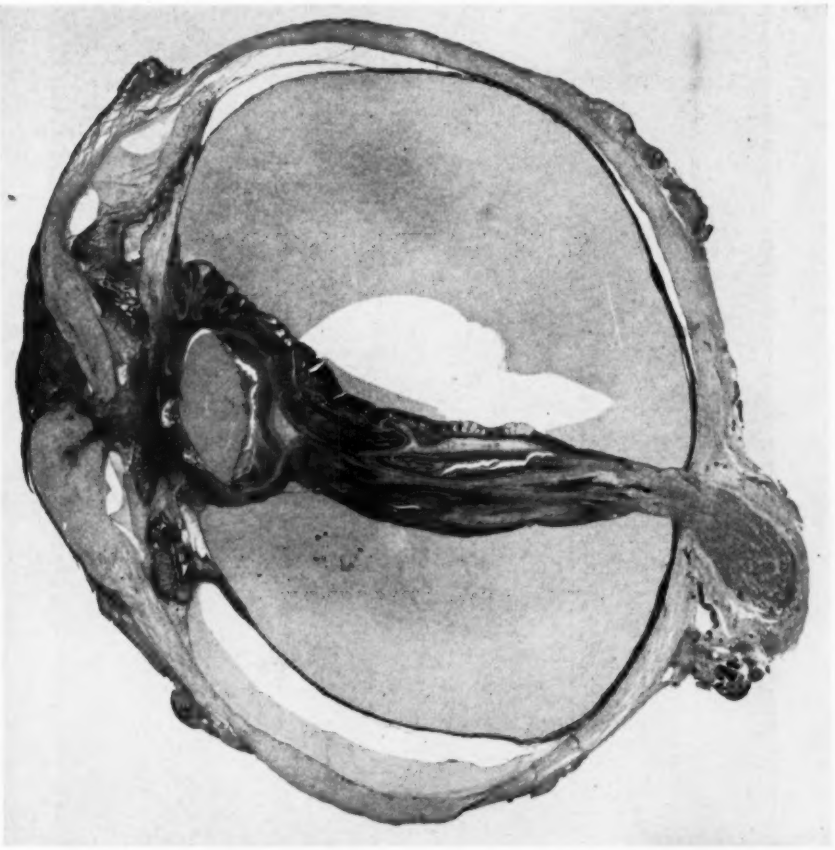

Fig. 3.—In this case of perforating injury, lens components escaped into the vitreous chamber (posterior rupture), as well as forward. The general appearance is that of a vitreous abscess bordered by detached retina.

retical sense. The "center" of the lesion may be an entire aqueous or vitreous chamber if lens substance has escaped into one of them (Figs. 2*a*, 3, and 4), or it may be a small sharply circumscribed region within the lens mass close to a site of rupture of the capsule (Figs. 5 and 6). The purulent exudate comes chiefly from the vascular tissues closest to the lens substance, where influence of the irritant (antigen) is most intense (Fig. 2*b*). If seen soon after the onset of the inflamma-

tion, the lesion would consist entirely of acute exudation from acutely inflamed vascular structures bordering the confines of the escaped lens substance. It would probably contain eosinophiles but otherwise be indistinguishable from an acute infectious lesion.

When the lesion is seen histologically, many weeks have usually elapsed, and more distinctive characteristics have developed. A zone of granulomatous tissue

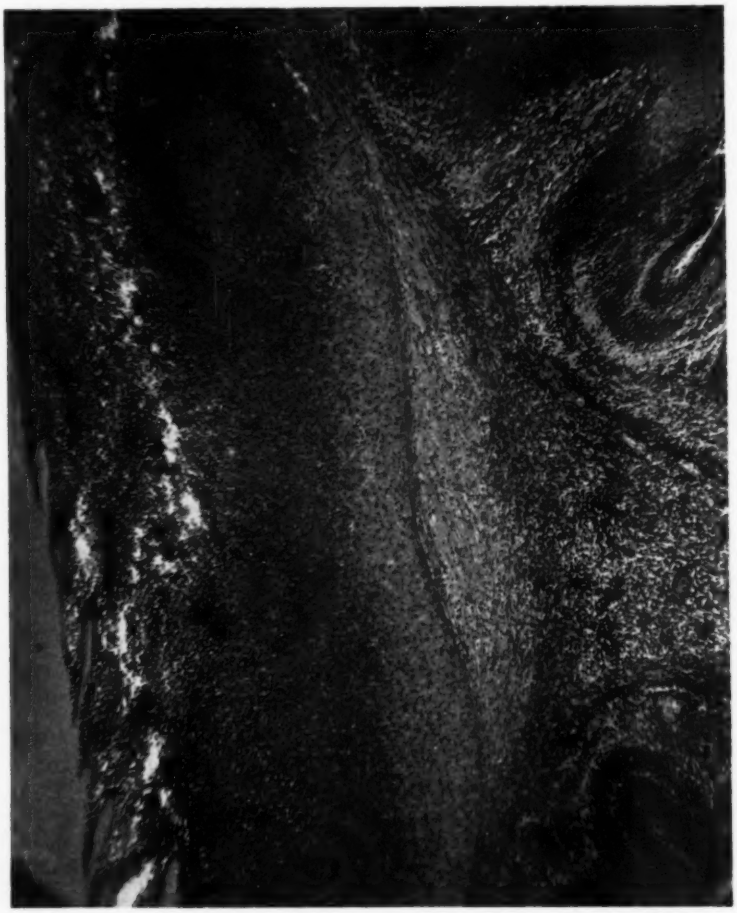

Fig. 4.—A portion of Figure 3 is shown in higher magnification. Disintegrating lens is seen to the left of a center of purulent exudate. A broad pale-staining band (down the center of the field) is an epithelioid zone that encloses the purulent focus. The triangular mass to the right is adventitious vascular connective tissue that unites folds of detached retina.

forms at the periphery of the purulent focus. It is produced by macrophages, some of them multinucleated giant cells, which undergo metaplasia into fixed epithelioid cells and acquire histic relations. Some influence, probably a chemotaxis, tends to line them up in a radial pattern, with their long axes pointing toward the purulent

center of the lesion. This is the granulomatous zone in the diagram. It may consist only of an arc that closes over a gap in the lens capsule (Fig. 5[7]), or it may partially, or even completely, line one or more of the chambers (Fig. 19). This tissue

Fig. 5. A pocket, partially enclosed by lens capsule, contains purulent exudate and disintegrating lens material. It is sealed anteriorly by an arc of fibrovascular tissue continuous with disorganized iris. An epithelioid border is interposed between the fibrous tissue and the purulent center. A cyclitic membrane covers the posterior (right) surface of the lens mass.

7. The reaction in this specimen centered on a site of degeneration of lens matter in an after-cataract in the form of a ring of Soemmerring. Bladder cells of Elschnig were sealed off in part of the ring and are more or less free in the area pictured. I have observed that "bladder cells," representing atypical globular proliferation of the capsular epithelium of the lens and commonly found in after-cataract lenses and other abnormal conditions, do not seem to play a role in production of lens reactions. This is probably true, for they are live structures with intact cell membranes. It might be expected that they would have to die and be decomposed, with liberation of their various fractions, before contributing any allergic or toxic influence.

contains many leucocytes, and along its inner border its cells mingle irregularly with those of the purulent mass. Its outer confines are, likewise, not sharply set off but merge into a vascular zone of ordinary fibroblastic tissue, which may fuse, for example, with the iris, ciliary body, or other ordinarily free surfaces (Fig. 6). While not usually an encircling one, this zone of fibrosis is viewed in the schematic lesion as being more remote from the center of irritation and as a later, more chronic,

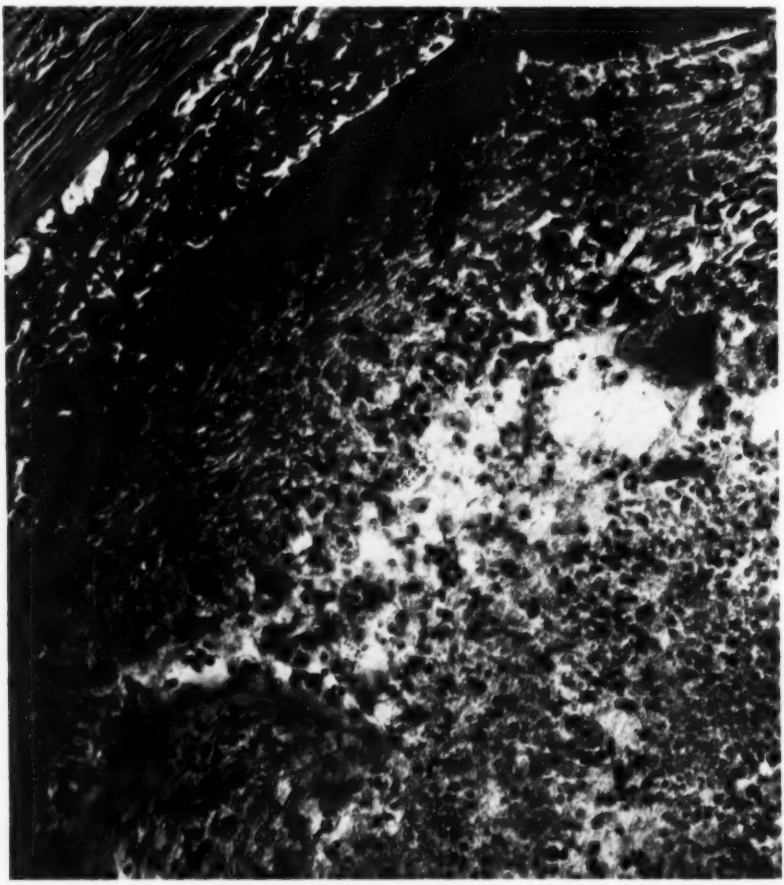

Fig. 6.—A higher magnification of a portion of Figure 5 shows details of the transition from acute purulent exudate (lower right corner) through a loose border zone of intermingled macrophages and pus cells to an epithelioid tissue layer, which merges into a vascular fibroblastic zone. The last-mentioned structure is fused with the posterior surface of the iris. Note the giant macrophages and compare with Figure 22.

aspect of the inflammatory reaction, comparable to the fibroblastic zone toward the periphery of an infectious abscess in a solid organ. Cyclitic membranes are regarded as belonging to the fibroblastic zone. Still more peripheral, in a figurative sense, is a zone of plasma cell infiltration. This zone in the well-established, older lesions

is almost always most pronounced in the iris and ciliary portion of the ciliary body (Fig. 21), but may involve also anterior portions of the choroid and the subepithelial limbic tissues. Regularly, it contains an admixture of scattered lymphocytes and other leucocytes.

A zone of lymphocytic infiltration is outermost. It represents the reaction in tissues most mildly irritated and ordinarily showing only small numbers of lymphocytes. In the retina and in the optic nerve head this is apt to be a perivascular reaction (Fig. 14). The fundal portion of the choroid is likely to be affected only by

Fig. 7.—The low magnification emphasizes the localization of the major pathologic changes in relation to the pupillary portion of the iris and the contiguous portion of the lens. There is also the generalized atrophy of glaucoma.

this minimal type of inflammatory change. The reaction in the subepithelial limbic region may also be of this type.

It must be kept in mind that part of what is seen in a particular case may be the result of ordinary, nonspecific reaction to trauma. However, a composite picture, drawn from correlation of the characteristics of many cases, of the disease, especially those in which there has been no trauma, will be that of a distinctive lesion, which, in my opinion, should now be recognized as a histopathologic entity whether or not it be considered a clinical entity, and regardless of doubts and conflicts concerning

its etiology and pathogenesis. Study of available textbooks and atlases of ophthalmic pathology would leave one almost unaware of the occurrence of such a lesion.

A variant of the pathognomonic picture is found when the process is of very long standing or, perhaps, has been particularly mild, so that the reaction immediately about the lens matter is more like an ordinary "foreign-body" granuloma. Such a reaction was shown in the case of a man aged 77 whose eye was enucleated because of persistent pain one year after a perforating injury (Fig. 7). Aside from gen-

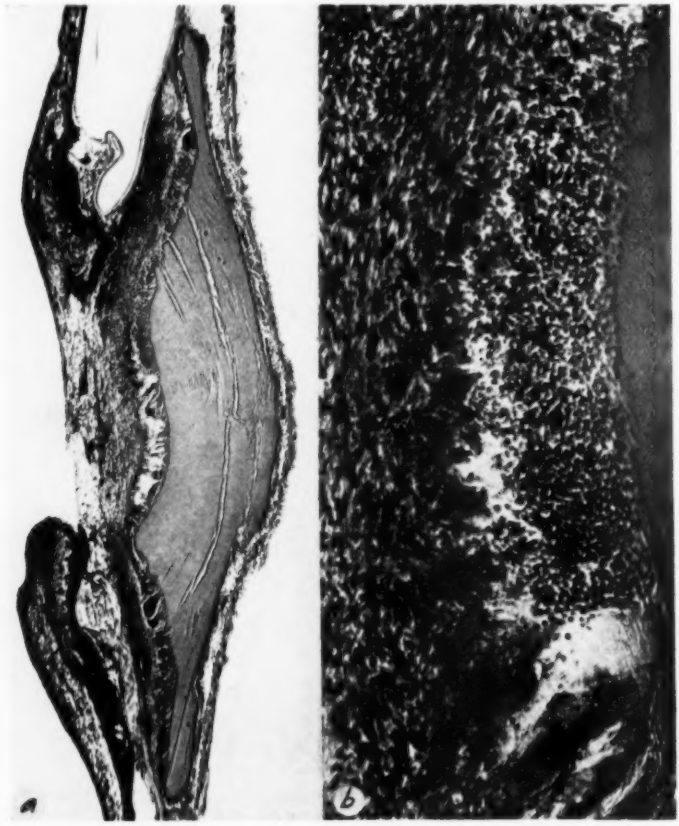

Fig. 8.—A higher magnification of the lesion shown in Figure 7 illustrates a particularly low-grade and chronic lens reaction, much of which resembles a foreign-body granuloma. (*a*) The moth-eaten remnant of nucleus is surrounded by vascular scar tissue. (*b*) A small purulent pocket is interposed between lens substance and an epithelioid border.

eralized glaucomatous atrophy, the only obvious abnormalities were limited to the pupillary region. No lens cortex structures remained, but there was an eroded nuclear remnant, about which giant macrophages formed an almost encircling layer (Figs. 8*a* and 9). At one point, however, a purulent focus was interposed between the lens substance and a fringe-like epithelioid zone (Fig. 8*b*). These figures illustrate strikingly the localization of major abnormalities to the structures close to a

site of liberation of the irritant, in this case a disintegrating lens nucleus. The presence of a characteristic lesion with purulent and epithelioid zones one year after the onset of the disorder is especially significant and suggests the advisability of getting rid of the lens matter in such cases as soon as possible. I believe the inflammation would have continued as long as any of the nuclear material remained.

REPORT OF CASES OF BILATERAL ENDOPHTHALMITIS PHACOANAPHYLACTICA

Two cases of bilateral endophthalmitis phacoanaphylactica diagnosed, in each instance, by examination of the first, or "exciting," eye will be presented in the

Fig. 9 (same eye as that shown in Figures 7 and 8).—The nuclear remnant, with its fibers fraying out to the left, is surrounded directly by macrophages, many of which are huge and multinucleated.

form of brief clinical summaries and of anatomic descriptions and illustrations that stress distinctive or unusual features. A third case, in which the eye secondarily involved was available for study, will be more completely detailed from the histopathologic standpoint, since it may prove to be the first unequivocal example of involvement of the "second" eye in the bilateral form of the disease to be reported as such.

Case 1.—B. E., a white woman aged 69, was under the care of Dr. Ralph I. Lloyd, who furnished the clinical data. The patient was suffering from a mental derangement and had been, until a short time before, in a psychiatric hospital. The right eye had become inflamed and painful, and examination in September, 1947, showed deposits on the posterior surface of the cornea and exudate in the pupillary area. A Vollmer skin test was strongly positive. The inflammation was controlled but flared up several times.

By January, 1948, the process in the right eye had decreased considerably, but the left eye became red and its tension rose. Despite the use of codeine and local miotics, the elevated tension

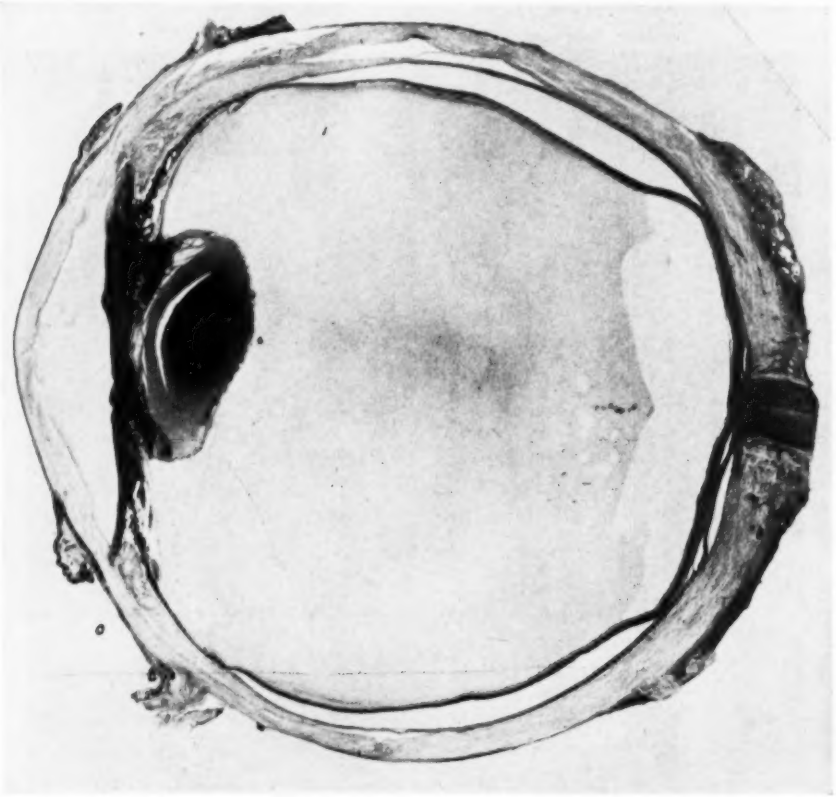

Fig. 10 (Case 1).—In a vertical meridional section, the lens, with a ruptured anterior capsule, is continuous with a darkly staining mass that occupies the iris, pupil, and posterior chamber. A cyclitic membrane can be seen, and the ciliary body (above) is partially detached.

and redness persisted. On Feb. 21, at the Brooklyn Eye and Ear Hospital, the right eye was enucleated, and a corneoscleral trephination was done on the left eye.

Tests for cutaneous sensitivity to lens and uveal pigment were done on March 25, and the results were interpreted as positive for both antigens. The site of injection of the pigment, however, was not excised for histologic examination.

The left eye had a shallow anterior chamber, from which blood was absorbed slowly. The tension remained elevated, and there was undue persistence of injection. A draining bleb had formed by the seventh day, and slow improvement had set in. Thereafter, the tension slowly

declined, and there were gradual reduction of chemosis and continued absorption of the hyphema until the time of discharge, on April 15.

When last examined, on May 5, 1948, the eye was described as showing hyperemia but was slowly becoming white. It was occasionally painful. The intraocular tension was 20 mm. Schiøtz. The anterior chamber was shallow; there was pigment on the lens capsule, and the iris was atrophic and adherent to the lens.

Fig. 11 (Case 1).—A portion of the purulent, epithelioid, and fibroblastic zones that lie anterior to the disintegrating lens substance is shown in detail.

Examination of the enucleated (right) eye revealed a rupture of the anterior capsule of the lens with an inflammatory reaction typical of endophthalmitis phacoanaphylactica centering on the site of rupture and to a large extent confined to the anterior segment (Fig. 10). Dr. F. H. Verhoeff concurred in the anatomic diagnosis.

Comment on Distinctive Anatomic Features.—Up to the time the histologic report was submitted, there was no knowledge of any injury to either eye, and no

evidence of trauma had been clinically detected. The patient's mental state was such that a reliable history could not be obtained. Inquiry later elicited the statement from others that the woman had injured her right eye by running into a door. However, there was no evidence of any perforating injury, so that exogenous infection can be excluded whether the rupture of the lens capsule is considered traumatic or spontaneous.

Fig. 12 (Case 1).—A cyclitic membrane, taking origin from an area of great distortion of nonpigmented and pigmented epithelium, contains fibroblasts and capillaries and a variety of leucocytes. This process is in no way distinguishable from cyclitic membranes in nonspecific inflammations.

The lesion, when enucleation was done, was of over five months' duration, and, although it was clinically relatively quiescent, there was still a purulent pocket containing disintegrating lens material and bordered anteriorly by an epithelioid fringe

(Fig. 11). A well-developed cyclitic membrane (Fig. 12) streamed forward to merge with fibrovascular tissues that walled off the anterior portion of the lens, obliterated the posterior chamber, and filled the pupil. The characteristic heavy leucocytic infiltration of the iris was predominantly plasmocytic but included occasional nodules of lymphocytes (Fig. 13). The ciliary body was partially detached,

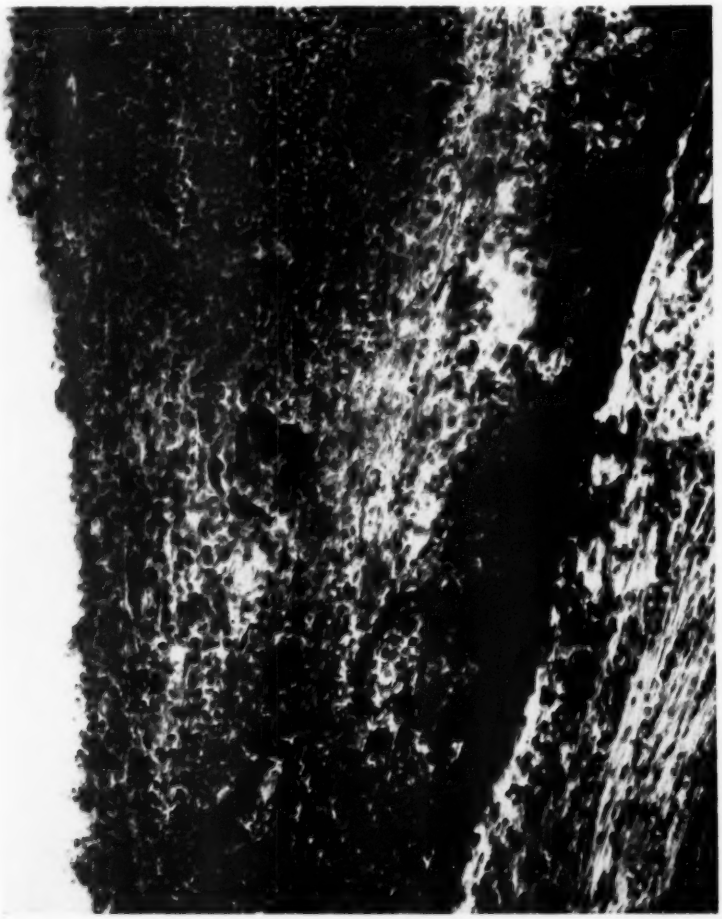

Fig. 13 (Case 1).—The iris, with its pigmented epithelium to the right, is heavily infiltrated with leucocytes, with a large component of plasma cells. At the top of the field is a densely packed lymphocytic nodule. Fibrovascular tissue adheres to the posterior surface of the iris.

so that atrophy of the bulb was inevitable. Choroidal inflammation was slight, but there was more than the usual involvement of the retina and optic nerve head (Fig. 14), with early formation of adventitious membranes reaching into the vitreous body (retinitis proliferans).

CASE 2.[8]—N. P., a white man aged 70, whose clinical history was obtained through the courtesy of Dr. Daniel Kravitz and Dr. John M. McLean, had been treated at a municipal hospital over a period of 24 years for chronic glaucoma of the left eye. When seen by Dr. Kravitz, on Nov. 4, 1946, the patient complained of sudden loss of vision, severe headache, and vomiting during the previous 24 hours. Both eyes presented the classic signs of acute glaucoma. Medication yielded no significant improvement; so the following day the patient was admitted

Fig. 14 (Case 1).—Inflammation in the retina and optic nerve head is more acute than usual, perhaps because of the severity of the disease and the relatively early stage at which enucleation was done. The degree of participation of tissue so remote from the lens seems to be an index of the amount of escape of lens antigen into the vitreous chamber.

to the Brooklyn Eye and Ear Hospital, where a basal iridectomy was done on the right eye and a modified Lagrange operation on the left eye.

8. This is the case referred to in a previous publication.[3]

After an uneventful convalescence, the patient was discharged from the hospital with a tension of 22 mm. in the right eye and of 20 mm. in the left eye (Schiøtz). Diffuse lenticular changes soon appeared in both eyes. By Jan. 9, 1947, tension in the right eye had increased to 2+, and the eye was considerably inflamed. Within three weeks it became stony-hard and presented the picture of severe plastic iridocyclitis. It was enucleated on Jan. 31. The left eye had been free of any signs of inflammation when its fellow was removed, and it was still white and had a tension of 20 mm. when examined seven days later. The patient was unable to return until Feb. 18 (18 days after the enucleation), when the remaining eye was painful and was found to be red, with elevated tension and many large deposits on the posterior surface of the cornea.

On Feb. 20, the man was admitted to the ophthalmologic service of Dr. John M. McLean at the New York Hospital, where he remained about six weeks. The diagnosis was "acute secondary

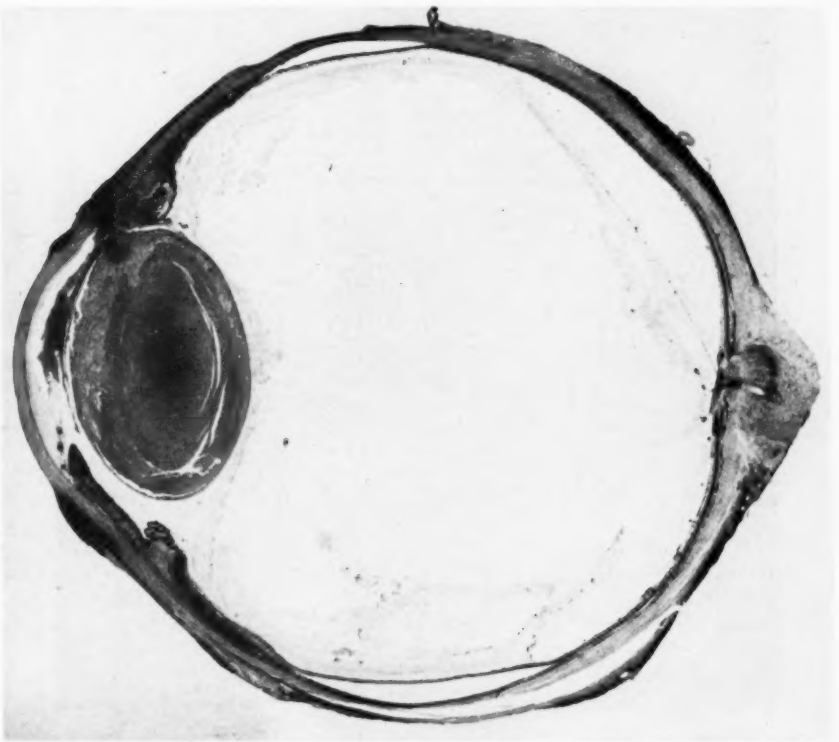

Fig. 15 (Case 2).—An intumescent, partially liquefied lens with a ruptured capsule has been drawn toward a limbic iridectomy wound (above). Widespread inflammation, most intense in the anterior segment, is apparent.

glaucoma of the left eye, due to chronic uveitis, and possible sympathetic ophthalmia of the same eye." Tension in the eye was reduced chiefly by paracentesis. At the time of discharge the tension had subsided sufficiently to be controlled by reopening of the paracentesis site once every two to three days. Further treatment was to be given at the clinic.

During this period of hospitalization, aqueous cell counts ranged from 284 to 2,000 per cubic millimeter, with 20% polymorphonuclear leucocytes and 80% lymphocytes. A test for sensitivity to uveal pigment was interpreted as negative, but sensitivity to lens antigen was evidently not tested. A Mazzini test (blood) was negative for tuberculosis, the colloidal gold curve of the spinal fluid was normal, and the Wassermann reaction of the spinal fluid was negative.

Examination of the enucleated (right) eye revealed an early, acute lesion of endophthalmitis phacoanaphylactica with its major localization in the anterior segment (Fig. 15). The lens was intumescent and partially liquefied. Its capsule had ruptured close to a limbic (iridectomy) wound.

Comment on Distinctive Anatomic Features.—In this eye, known to have been inflamed for only 22 days, acute exudative inflammation predominated, while the only appreciable proliferative (productive) features were of a granulomatous character. Fibrous adhesions and masses of fibrous and vascular new tissue had not yet made their appearance. The inflammatory reaction, in the absence of effective

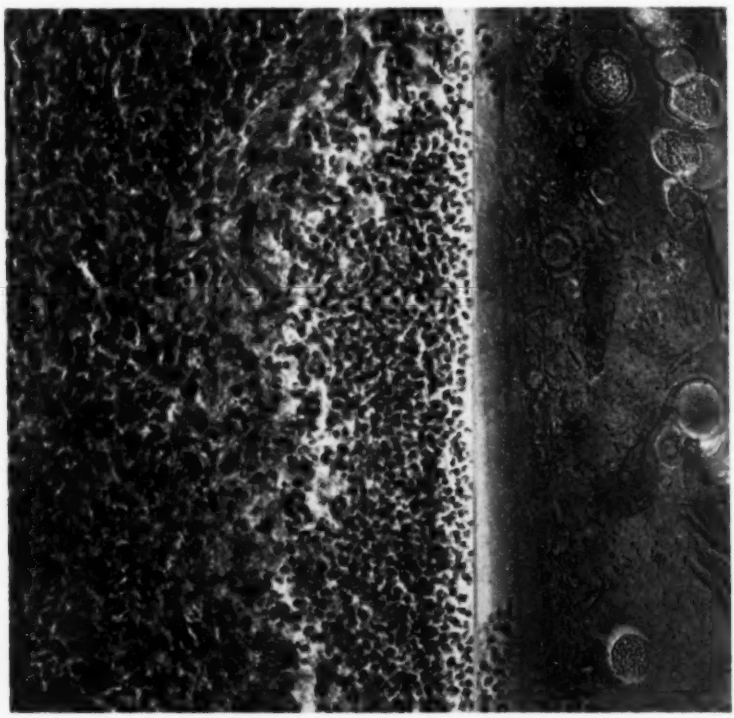

Fig. 16 (Case 2).—Anterior to the lens capsule, a purulent pocket is bordered by a ragged wall of macrophages and epithelioid granulation tissue, which is becoming transformed into ordinary vascular and fibroblastic connective tissue. The latter is rich in leucocytes of mixed composition. On the right the lens mass contains homogeneous (gray) liquefied lens matter, dissociated fibers, and Morgagnian globules. The picture has been reconstructed to eliminate an artificial cleft.

"walling off," and probably because of the great intensity of the irritation, was of a more generalized nature than usual, involving all vascular tissues. There was, however, a much more acute reaction in the anterior structures than elsewhere, and the only purulent focus was in the anterior chamber, into which the lens had ruptured. An early epithelioid layer of tissue was forming about the exudate, tending to enclose it (Fig. 16). In the region of the ora serrata several discrete Dalén-Fuchs nodules

were present, although there was nothing pathognomonic of sympathetic ophthalmia, the condition with which they are oftenest identified (Fig. 17).

CASE 3.—P. S., a man aged 65, was first seen on Oct. 1, 1947, by Dr. Benjamin C. Rosenthal, who furnished the clinical data. His complaint was of failing vision. He had suffered lime burns of the eyes many years before, after which there had been visual impairment. He had been told by several ophthalmologists that he had glaucoma and had been using miotics for the preceding

Fig. 17 (Case 2).—More than the usual leucocytic infiltration is seen in the choroid. A well-defined Dalén-Fuchs nodule has displaced the pigmented epithelium. The retinal separation is an artifact.

six months. Vision of 20/40 in the right eye and "shadows at 6 in." in the left eye could not be improved with glasses. Tension was elevated in both eyes.

An iridencleisis was performed on the right eye at the Brooklyn Eye and Ear Hospital on Oct. 23. After an uneventful recovery, the tension remained within normal limits until February,

1950. Changes in the anterior portion of the lens had begun six months after the operation and had progressed to the point where vision was reduced to 10/200 and could not be improved with glasses.

On Feb. 14, 1950, or 30 months after the iridencleisis, cataract surgery was undertaken. Because of the filtering cicatrix superiorly, it was decided to employ a temporal limbus flap and a keratome section. The iris was found to be adherent to the lens; so the lens was delivered extracapsularly. The postoperative reaction was severe, with the anterior chamber remaining shallow for more than two weeks; lens absorption was delayed. Five weeks after operations, a secondary cataract was observed; the eye was quiet, but the tension was lower than normal.

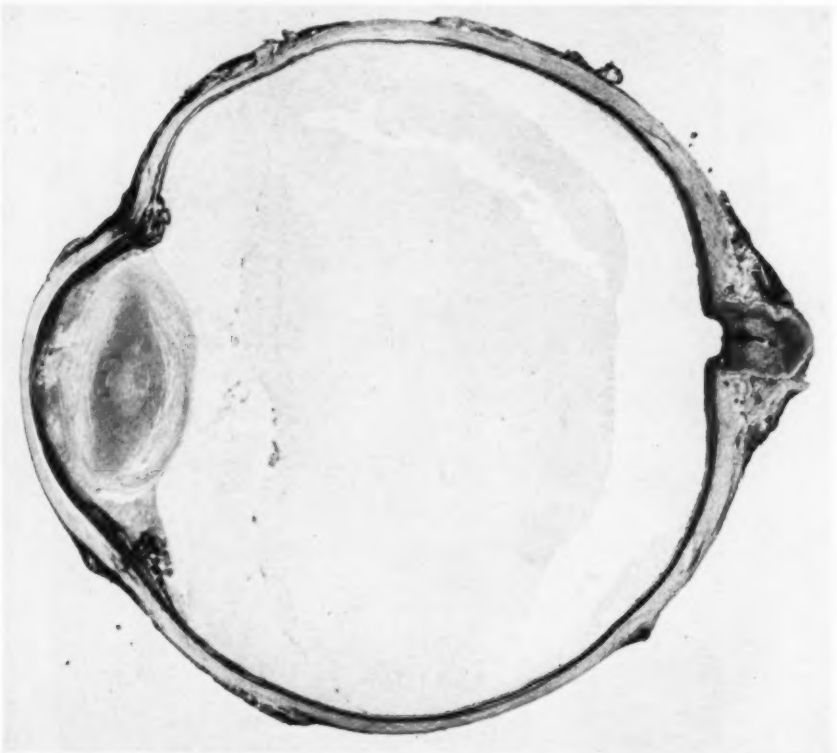

Fig. 18 (Case 3).—The eye was secondarily involved in a case of bilateral endophthalmitis phacoanaphylactica. The anterior chamber has been obliterated by prolapse and total adhesion of the iris to the cornea. The lens capsule has ruptured into the posterior chamber. Widespread inflammation has its major localization in the anterior segment. There is well-established glaucoma.

Seven weeks after operation hypopyon developed in the right eye. The cornea was vascularized temporally. The iris was also somewhat vascularized, and the anterior chamber showed a pronounced flare. A diagnosis of uveitis or vitreous infection was made, and antibiotics, cortisone, and other indicated medication were employed. The pericorneal injection and hypopyon increased, necessitating paracentesis. Intravitreous injection of penicillin was also used. The inflammation gradually quieted down. A small fistula then appeared at the limbus temporally. Three months after the cataract operation, there was evidence of beginning phthisis bulbi. Four months after operation, there remained no light perception, the eye was shrunken, and the tension was —2.

The left eye retained light perception only. It was quiet, and the anterior chamber remained shallow, but the tension remained above 3⅝ mm. (Schiøtz). There were no precipitates or cells in the anterior chamber.

On July 25, 1950, five and one-half months after the cataract operation, the patient was knocked down by an automobile, and immediately afterward pain developed in the left eye.

Fig. 19 (Case 3).—The posterior chamber into which lens material escaped has become filled with acute exudate and has acquired a granulomatous lining, which is shown covering the back of the iris and the anterior surface of the ciliary body. Some lens derivatives are visible in the upper right corner.

Examination six days later showed that the right eye was quiet and atrophic. The left eye presented pericorneal injection, a steamy cornea, a dilated pupil, and many large precipitates of the mutton-fat variety. The eye was stony-hard. There was no evidence of traumatic ocular perforation.

Dr. Rosenthal was of the opinion that the patient had acute uveitis, probably sympathetic ophthalmia. The left eye became more painful; but there was some response to the administration of cortisone, antibiotics, and other medication, so that enucleation was not then advised. For a few months the condition seemed to improve and the patient was comfortable, but about seven months after the injury he manifested evidences of beginning corneal degeneration, with iridic hemorrhages and pain. Enucleation was performed on May 22, 1951, one year and three months after cataract surgery on the fellow eye. At that time the right eye was not inflamed. The

Fig. 20 (Case 3).—Another portion of the posterior chamber is occupied by acute purulent exudate and enveloping epithelioid and fibrovascular layers, which are continuous with inflamed uveal tissue.

enucleated (left) eye showed a rather acute form of endophthalmitis phacoanaphylactica associated with anterior rupture of the lens capsule. The eye was also the seat of chronic glaucoma (Fig. 18).

Comment on Distinctive Anatomic Features.—The left eye became inflamed immediately after injury, and five and one-half months after a postoperative inflam-

matory episode in the right eye, at a time when the process in the right eye was quiescent. The histologic characteristics were in keeping with the history of disease in the second eye of about 10 months' duration.

The anterior chamber had been obliterated by prolapse of the iris and total adhesion to the cornea. Lens matter had escaped into the posterior chamber, and it was

Fig. 21 (Case 3).—Plasma cells and other leucocytes are seen in the ciliary body. Macrophages, with an occasional giant cell, on the surface have acquired granulomatous characteristics.

chiefly into that space that acute exudation occurred. The chamber had become lined by a granulomatous wall enclosing purulent exudate (Figs. 19 and 20). There was, however, more than the usual participation of vascular tissues bordering the vitreous body, where large giant cells were occasionally seen among macrophages and other leucocytes (Figs. 21 and 22).

The lesion present in this second, or "sympathizing," eye might be called a "pure" lesion in the sense of not being complicated by any recognizable direct results of trauma, unless the rupture of the lens capsule is considered traumatic. It is noteworthy that the process was otherwise identical with that found in the unilateral form of the disease.

Fig. 22 (Case 3).—Newly forming granulomatous tissue lines part of the surface of the retina and the optic nerve head. This type of involvement, not uncommonly encountered, is interpreted as part of zone *B* in the schematic lesion (Fig. 1).

SUGGESTIONS FOR DIAGNOSIS AND THERAPY

For the pathologist to advise the clinical ophthalmologist on diagnostic and therapeutic procedures may seem gratuitous; but the following suggestions, based chiefly on pathologic experience, are ventured in the hope that somewhat fewer clinical problems in this category will have to be solved by examination of enucleated, sightless eyes.

1. Skin testing for sensitivity to lens protein (and uveal pigment also) in cases of unexpectedly severe or prolonged postoperative or post-traumatic uveitis may yield valuable information. This should be done whether or not the lens capsule is known or thought to be defective.

2. If the skin reaction to lens protein is positive, it would seem wise to follow Courtney's suggestion and get rid of the lens in the inflamed eye as promptly and as completely as possible, without waiting for the condition to "quiet down." If a disintegrating lens is present, I should expect that attempts at desensitization would fail to control completely the inflammation.

3. In cases of unilateral endophthalmitis phacoanaphylactica following extracapsular cataract extraction, desensitization is a logical form of therapy, with value also, perhaps, as prophylaxis against "spontaneous" involvement of the other eye, and it should certainly be employed prior to operation on the other eye. Subsequent cataract extraction should be intracapsular if possible; otherwise lens material should be eliminated as completely as possible.

4. Enucleated inflamed eyes submitted to the pathologist should be accompanied by enough of the history to inform him of any possibility of endophthalmitis or sympathetic ophthalmia, so that such specimens may be given preferential treatment and reported on promptly.

5. The pathologist's report of endophthalmitis phacoanaphylactica in the enucleated eye of a patient whose second eye has become inflamed probably means that the second eye has a defective lens capsule. Prompt removal of lens matter, as complete as possible, would seem to be indicated, rather than an attempt, by intradermal injections of dilute lens antigen, to render the ocular tissues nonreactive to the continuing release of an irritant, in high concentration, from the patient's own disintegrating lens.

6. The development of inflammation in a second eye with a simultaneous or slightly preceding exacerbation of activity in the eye first involved speaks for the presence of true sympathetic ophthalmia rather than bilateral "lens reaction"; in such instances the value of having tested for sensitivity to uveal pigment will be apparent.

SUMMARY AND CONCLUSIONS

The varied anatomic characteristics of the lesion called endophthalmitis phacoanaphylactica, lens-induced endophthalmitis, or lens reaction are analyzed and described in detail. The belief is advanced that the disorder should become generally accepted as an important histopathologic entity.

Even though there is lack of complete understanding of its etiology and pathogenesis, it seems certain that allergy constitutes at least an important aspect of the disease, as evidenced by its histopathologic features and by sensitivity to lens protein demonstrable by skin testing.

In the belief, with others,[9] that there is a bilateral form of the disease which may be confused with sympathetic ophthalmia, three cases of bilateral ocular inflammation are presented, with special emphasis on the anatomic characteristics of one enucleated eye in each case.

9. Footnotes 2 and 4.

In two cases the eye first involved in inflammation was enucleated and found to have a ruptured lens capsule. It was not ascertainable, in either case, whether the lens of the remaining (secondarily involved) eye had an intact or a ruptured capsule, but in each case that lens had been cataractous. In a third case the eye secondarily involved was enucleated and its lens capsule was found ruptured. In this instance inflammation in the first eye had followed an extracapsular cataract extraction. These anatomic findings support the belief that rupture of the capsule is usually, and perhaps invariably, present in the one eye in cases of the unilateral form and in both eyes in cases of the bilateral form.

In only one of the three cases had there been any perforating injury (surgical or accidental) of the eye that was enucleated. In that instance a basal iridectomy had preceded the inflammatory process. In the other two cases there was a history of some injury, but the eyes presented neither clinical nor histologic evidence of direct violence, and certainly no perforating wound. These two specimens were, in any case, free of direct results of perforation of the bulb and free of exogenous infection. They can, therefore, be regarded as showing the lesion under discussion in an uncomplicated, or pure form. One of the two was the eye first involved in a case of bilateral disease; the other was the "second" eye in another case. The histologic lesions found were identical in all essential respects.

There had been a test for skin sensitivity to lens protein in only one of the three cases discussed, and the reaction to that was positive. In the other cases, skin testing might have furnished valuable information before enucleation and anatomic diagnosis of the condition.

Study of many cases of unilateral endophthalmitis phacoanaphylactica with persistence of inflammation about lens remnants long after the onset of the disease suggests that the process will continue as long as lens substance remains.

Various suggestions concerning diagnosis and therapy are outlined: they include more frequent and earlier tests for sensitivity to lens and pigment antigens, early removal of the lens of the inflamed eye if the patient is skin-reactive to lens protein, and an attempt at desensitization when the lens of the inflamed eye has been extracted by the extracapsular method or when operation on the other eye is contemplated. In bilateral cases, if either eye has been enucleated, histopathologic examination should be promptly reported. This information, combined with the results of skin tests, should in most instances clearly differentiate lens-induced endophthalmitis from sympathetic ophthalmia. Earlier diagnosis and appropriate therapy of either condition may lead to earlier arrest of the inflammation and greater salvage of vision.

FOCAL SCLERAL NECROSIS

A Late Sequel of Irradiation

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AND
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NEW YORK

RADIATION therapy about the eye is being employed by an increasing number of practitioners in an expanding category of conditions. It is only to be expected that an increase in the late complications of irradiation will be experienced. Radiation cataract, the commonest late complication, is known to every ophthalmologist. Its clinical and pathological characteristics have received a great deal of attention. In like manner, late skin changes and effects on bone growth are well-known and easily recognized sequelae. The purpose of this paper is to report a less common late effect of application of radiation to the globe which presents a striking clinical appearance, namely, focal scleral necrosis. So far as could be determined, this sequela has not previously been described.

PRESENTATION OF CASES

CASE 1.—J. G., a white man aged 38, had had a squamous-cell carcinoma of the right lower lid treated 18 years earlier with radium, 400 mg.-hr. being given over a period of 14 weeks; the other factors were unknown. Twenty-six months later he exhibited a growth on the conjunctiva of the right eye in the area exposed by the lid defect. This growth was excised and reported as being a papilloma with epitheliomatous changes. The area was treated with radiation, but the dosage is not known. In the interval, the patient was symptom-free until one month before admission, when a "recurrent" growth appeared at the site of the original tumor. On examination a sharply demarcated, punched-out hole, measuring approximately 1.5 mm. in diameter, was seen in the sclera of the right eye near the limbus at 8 o'clock. In the floor of this hole uveal pigment could be seen. The area surrounding the hole was blanched. Tension was 14 mm. (Schiøtz) in this eye and 18 mm. in the opposite eye. Vision in the affected eye was 20/30. The lateral three-quarters of the lower lid showed loss of the lid margin. Very little cul-de-sac was present, and the bulbar conjunctiva corresponding to the defect in the lid had undergone keratinization. Figure 1 shows the clinical appearance of the affected eye, with a sharply demarcated hole in a white, avascular scleral area. The scleral dehiscence was covered by a conjunctival flap, and the patient made an uneventful recovery.

CASE 2.—W. R., a white man aged 72, had a local excision of a squamous-cell epithelioma of the temporal bulbar conjunctiva of the right eye one month after it was noticed. Seven months later a growth, apparently recurrent, in the same spot was treated with eight one-minute surface applications of beta radiation over a 35-day period. The strontium applicator used supplied 11,000 rep per hour at the surface, or a total dosage of less than 1,500 rep. Eight days after the last application an ulcer developed at the treatment site. The ulcer was a round, deep, punched-out hole in the sclera, and uveal tissue was not noted in its floor. Healing occurred after a conjunctival flap had been drawn over the hole, but over the next few months the conjunctiva

From the Institute of Ophthalmology, Presbyterian Hospital.

Fig. 1 (Case 1).—Punched-out scleral hole (*a*); avascular area (*b*).

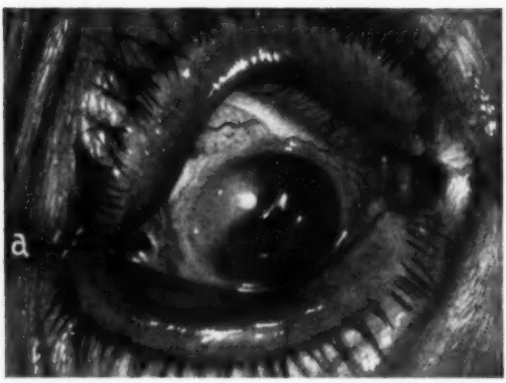

Fig. 2 (Case 2).—Punched-out scleral hole (*a*).

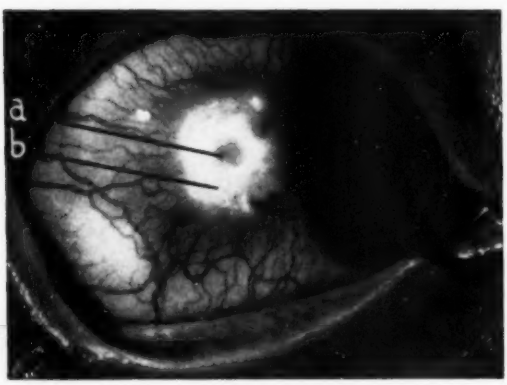

Fig. 3 (Case 3).—Punched-out scleral hole (*a*); avascular area (*b*).

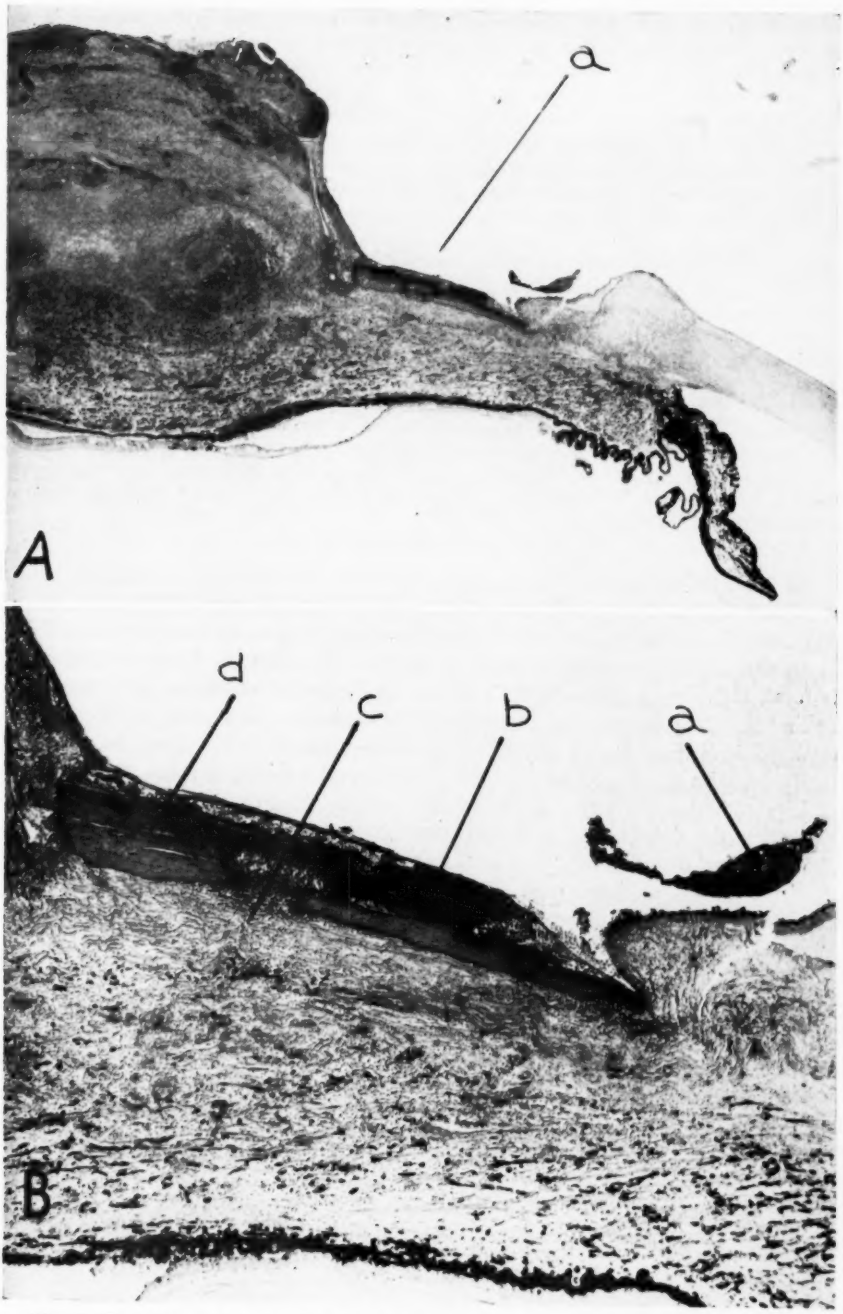

Fig. 4 (Case 3).—*A*, low-power view of the eye: crater (*a*). *B*, high-power view: slough (*a*); sequestrum (*b*); fragmented scleral tissue (*c*); calcified sclera (*d*).

ulcerated, healed, and scarred progressively. Eighteen months after first appearance of the growth, it became apparent that an extension of the epithelioma into the orbit had taken place. Figure 2 shows the appearance of the globe at this time. Near the temporal limbus can be seen the punched-out hole resulting from scleral necrosis. The adjacent cornea is cloudy, and the surrounding conjunctiva shows diminished vascularity. Exenteration of the orbit was carried out.

Case 3.—L. R., a white man aged 72, had had a carcinoma of the limbus of the right eye treated with radium four years previously. The total dose was 1,000 mc.-min., the other factors being unknown. Within one month there developed a white, avascular area at the site of the treatment. Four years after treatment an ulcer developed in the center of this avascular spot. The situation was complicated by secondary glaucoma. On examination, the pearly-white area which had developed at the site of irradiation was still present, and in the center of it was a round, ulcerated, sharply defined hole. Since the patient did not have useful vision in the eye, an enucleation was performed. Figure 3 shows the clinical appearance of the area of scleral necrosis. Although the lesion is not as clear as in case 1, there can still be seen a large white, avascular area with a deeper ulcerated, necrotic area in the center. Figure 4*A* is a low-power view of a section through the ulcer. The excavated area, with slough in the cavity, is clearly shown. On the scleral side the tissue is swollen as a result of necrosis, edema, and cellular infiltration. Figure 4*B* is a high-power view of the same area. On the scleral side fragmentation and necrosis of the scleral fibers can be seen, together with cellular infiltration. Just beneath the floor of the ulcer is a calcified structure, which appears to be a piece of sclera. This apparently is in the process of sequestration. A few scleral fibers still remain between the floor of the ulcer and the choroid.

COMMENT

It is commonly known that bone may undergo necrosis after irradiation, and it may well be that the condition reported in these cases is analogous, since the sclera, like bone, is relatively avascular. The sequestration phenomenon, as shown here, is another point in common with bone necrosis. The necrosis in these three cases is focal, following a concentrated localized application of radiation over the sclera or limbus. Both gamma and beta radiation are demonstrated to be capable of producing scleral necrosis. It may be worth noting that 18 and 4 years elapsed after treatment with gamma radiation, while only 6 weeks passed between the initiation of beta radiation therapy and the appearance of an ulcer. Nothing can be said regarding the dosage except that in these cases a minimum of 1,500 rep, with the other factors unknown, was sufficient to produce the necrosis. This is a beta dose considered moderate and commonly employed. The youngest patient here reported remained asymptomatic for the longest period after irradiation. Individual susceptibility would, no doubt, further modify the figures.

SUMMARY

Three cases of focal scleral necrosis, an unusual late complication of focal application of radiation to the sclera or limbus, are presented. The close analogy of this phenomenon to bone necrosis after irradiation is pointed out. The possibility of focal scleral necrosis should be considered when radiation to the sclera or limbus is contemplated.

73 E. 71st St. (21).

Dr. Daniel C. Kirby granted us permission to publish the second case.

PERIMETRY WITH ULTRAVIOLET (BLACK) RADIATION AND LUMINESCENT TEST OBJECTS

A Preliminary Report

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ONE OF the most difficult problems in perimetry has been the elimination of distraction in the examination. Test-object carriers; the examiner's hand and clothing; light reflected from marking pins, chalk, and perimeter arcs, and extraneous room light, all distract the patient's attention from both the fixation target and the test object in the peripheral field, thus adding to the difficulty of performing the examination and detracting from its accuracy.

The examination of the visual field by perimetry is a subjective test. It is the patient's interpretation of what he sees that determines the accuracy and validity of the test, and it is the visualization of the test object in relation to the fixation target that determines the extent of the visual field as interpreted by the individual patient. Any method which simplifies the test for the patient by eliminating distracting stimuli from within the field of vision so that only the fixation target and the peripherally located test object are visible will enhance both the accuracy and the ease of performance of the examination.

The numerous devices which have attempted to provide for this objective are mute testimony to its importance. A few of these may be mentioned: (1) the use of nonreflecting surfaces of black or gray felt on tangent screen or perimeter arc; (2) wearing of black gowns and black sleeves and gloves by the examiner; (3) projection of the target as a spot of light on the tangent screen or perimeter arc.[1]

Of these methods only the last successfully eliminates the distracting elements usually encountered in the examination.

All these problems are practically and economically solved through the use of "black light" and luminescent test objects in examination of the visual field with the perimeter and tangent screen. With this method distracting elements are almost completely eliminated from the field, only the fixation target and the test object being visible to the patient. Total darkness is not required, and the patient's fixation and reactions may be constantly observed by the examiner. The equipment required is simple and inexpensive and, in the case of the tangent screen, utilizes existing and commonly used materials.

From the Division of Ophthalmology, University of California School of Medicine.

1. Evans, J. N.: The Perimeter Spot Light Object, Arch. Ophth. **7**:614 (April) 1932.

The Harrington Black Light Perimeter and Lumilamp Tangent Screen Light used with the Harrington Lumitest Objects constitute the first practical application of ultraviolet radiation and luminescent test objects in the examination of the visual field. Both the Black Light Perimeter and the Lumilamp Tangent Screen Light emit ultraviolet rays below the visible spectrum.

The source of ultraviolet radiation for the perimeter is a cold cathode tube of 18 mm. diameter, rigidly suspended slightly above and 1½ in. (3.8 cm.) in front of the face of the perimeter arc. The tube parallels the perimeter arc for its full

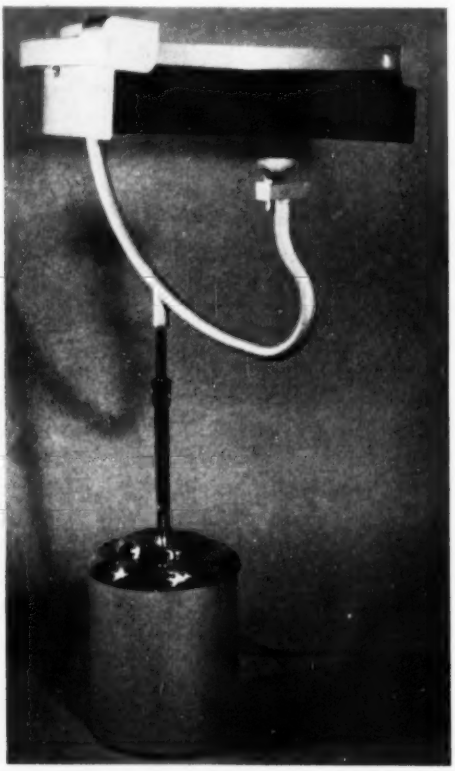

Fig. 1.—Perimeter, front view.

length, so that the intensity of the radiation source remains constant through the entire 180 degrees.

The tube contains B-10, a commercial conducting gas whose components are argon, neon, krypton, and helium plus a minute quantity of triple-distilled mercury. When voltage is applied to the tube electrodes, the B-10 gas is ionized, and the resultant heat vaporizes the mercury. The ultraviolet radiation thus generated by the ionized B-10 gas and vaporized mercury is amplified by ultraviolet radiation-emitting phosphors with which the inner wall of the tube is coated. As only a portion of the ultraviolet radiation produced by the tube is within the band useful for

visual field examination, the glass of the tube is tinted to a shade which filters out all the radiation except that between 320 and 380μ. The peak of radiation is at 365μ. The visible spectrum begins at 387μ.

The light tubes are standardized so that each tube emits the same number of lumens per foot directed against the surface of the perimeter arc with an even intensity over the entire 180 degrees. The estimated life of the tube is 15,000 hours, and the tubes are replaceable.

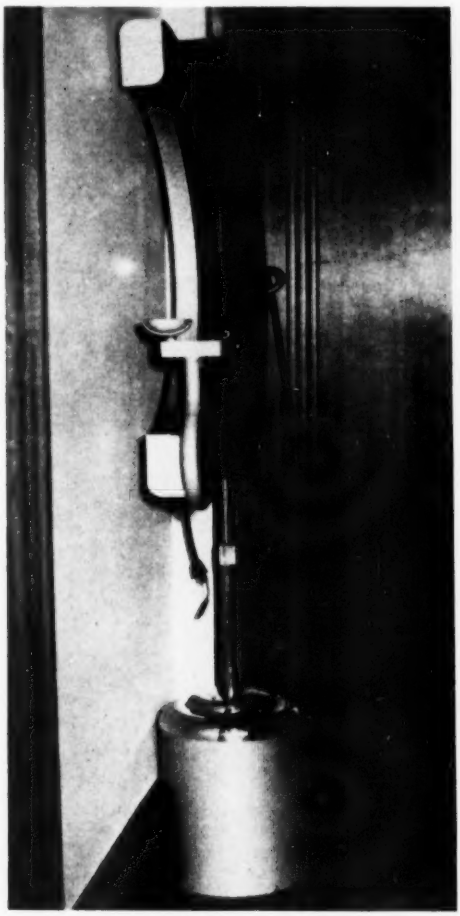

Fig. 2.—Perimeter arc in vertical position for storage.

The Lumilamp Tangent Screen Light employs a similar, but much smaller, radiation source, mounted in a convenient hand-held housing. The examiner directs its beam against the screen so as to illuminate the fixation point and the test object at the same time. The portability of the radiation source makes it possible to control the illumination at all times and to vary its intensity at will for greater sensitivity in the test.

The Lumitest Objects are printed in luminescent sulfide ink, which, when exposed to the ultraviolet radiation of the perimeter or the tangent-screen lamp, absorbs the "black light" quantitatively and transforms this invisible radiation into a white, red, green, or blue color, depending on the sulfide used. There is no heat, and the amount of visible light released by the targets when excited by the "black light" is relatively high.

The amount and type of luminescent sulfides used in the test objects are carefully controlled so that each test object will release the identical amount of light of exactly

Fig. 3.—Tangent screen lamp.

the same wave length as its corresponding master test object, maintained as a control by the manufacturer.

To heighten the contrast between the test object and the background and to eliminate the possibility of any ultraviolet radiation reaching the patient's or the examiner's eyes, the face of the perimeter arc and all surfaces of its base and supports are painted with enamels containing an ultraviolet radiation-absorbing compound. The felt of the tangent screen is sprayed with a colorless solution of the same ultraviolet-radiation absorber.

Since all the illumination from the radiation source is below the visible spectrum except for that light transformed by the test objects into visible rays of white, red, green, or blue, the test objects themselves appear to the patient as the "source" of the light, rather than as the reflectors of remote illumination. All sources of distraction fade into blackness, and the test objects become dots of pure light viewed against a perfect void.

Fig. 4.—Lumitest objects, printed in multiple sizes in luminescent sulfide pigments, in white and color. Individual test objects are to be cut from the strip and inserted in the slit in the black wand. They may be discarded when soiled.

Without the confusing reflection of light from the surface of the perimeter arc, tangent screen, test-object carrier, test object pins, etc., a 0.5 mm. white Lumitest object may be quickly and accurately detected by the patient the moment it enters the limits of its isopter, and it is quite possible to delimit sharply a minute scotoma in the central field with a 0.25 mm. test object at a distance of 2 meters, a feat impossible to duplicate with reflected light scotometry except with the most intelligent and observant patient.

The Harrington Black Perimeter Light is manufactured with either ultraviolet or visible radiation illumination. If standard illumination is desired for use with standard reflecting test objects, the cold cathode tube is coated so as to emit a white light of 450μ with an intensity of 7 foot-candles over the entire perimeter face. The standard spherical test objects of Berens [2] may be used with this instrument.

The instrument satisfies the primary requirements of a practical arc perimeter and possesses the following improvements over existing models:

1. It has an arc the radius of curvature of which is 330 mm. and which is of sufficient width to simulate a portion of a hollow sphere and thus allow for a degree of scotometry within the confines of this width without rotation of the arc.[3]

2. Illumination is uniform and constant on all portions of the arc.

3. The arc and the illuminating system are mounted on an adjustable microphone stand, thus eliminating the need of a table and allowing for easy adjustment to varying heights for the patient's greater comfort.

4. The surface of the perimeter arc is covered with felt or with ultraviolet radiation-absorbing, dull-black paint, depending on the model used, to minimize reflection.

5. The illuminating tube is parallel to the perimeter arc, so that when the arc is in the vertical position the entire instrument requires a minimum of storage space.

6. The weighted base is mounted on small casters so that the instrument may be wheeled from one room to another and does not require a table. It does not require a separate "perimeter room" for its use.

7. Standard Berens test objects [2] may be used with the white-light perimeter. A complete set of Lumitest objects with a wand painted with a dull-black, ultraviolet radiation-absorbing compound to carry the objects is included with the Black Light model.

8. The instrument is less expensive than most comparable models currently on the market.[4]

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2. Berens, C.; Kern, D., and Evans, J.: White and Colored Spherical Test Objects for Use in Campimetry and Perimetry, Tr. Sect. Ophth., A. M. A., p. 407, 1931.

3. Traquair, H. M.: An Introduction to Clinical Perimetry, Ed. 3, St. Louis, C. V. Mosby Company, 1938.

4. The Harrington Perimeter, Black Light Perimeter, Lumilamp Tangent Screen Light, and Lumitest Objects are manufactured by Jenkel-Davidson Optical Company, 366 Post St, San Francisco.

OPHTHALMOPLEGIC MIGRAINE

Pathogenesis; Report of Pathological Findings in a Case of Recurrent Oculomotor Paralysis

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AND
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OPHTHALMOPLEGIC migraine was first defined by Charcot[1] in 1890 as a "condition associated with migraine in which there is a palsy of an ocular nerve, usually the third, temporary at first and later becoming permanent."

This clinical syndrome is characterized by paroxysmal, recurrent, transient partial or total ophthalmoplegia associated with severe hemicrania and with or without scintillating scotoma, homonymous hemianopsia, depression, general malaise, nausea and vasomotor disturbances. It frequently progresses to total and permanent unilateral oculomotor paralysis.

Numerous individual variations and atypical paralyses were adequately reviewed by Alpers and Yaskin,[2] but the patient with typical ophthalmoplegic migraine presents the following clinical picture:

1. There is a history of typical migraine attacks, without ocular muscle paralysis, of many years' duration, prior to the first attack of ophthalmoplegia.

2. The individual attack may begin, as in any other form of migraine, with scintillating scotoma and homonymous hemianopsia, followed by severe hemicrania, nausea, and vomiting.

3. The headache and general malaise in ophthalmoplegic migraine are usually much severer and more protracted than in other forms and may last from a few hours to many weeks. The headache is a true hemicrania.

4. The ophthalmoplegia may occur during or after the hemicrania and usually involves only the oculomotor nerve, although instances of trochlear and abducens paralysis have been observed.

5. When the third nerve is involved, it is the peripheral portion that is affected, and invariably on the same side as the hemicrania. Rare instances of "alternating ophthalmoplegia" have been reported,[3] but in any given case the paralysis almost always occurs on the same side.

6. The paralysis becomes severer with each successive attack, until it is a permanent unilateral, usually total, oculomotor ophthalmoplegia.

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1. Charcot, J. M., cited by Rea, R. L.: Neuro-Ophthalmology, St. Louis, C. V. Mosby Company, 1938.

2. Alpers, B. J., and Yaskin, H. E.: Pathogenesis of Ophthalmoplegic Migraine, A. M. A. Arch. Ophth. **45**:555 (May) 1951.

3. Fry, W., and Schlezinger, N. S.: Variable Ophthalmoplegia Due to Angioneurotic Edema, A. M. A. Arch. Ophth. **46**:655 (Dec.) 1951.

The reported high incidence of recurrent oculomotor paralysis associated with intracranial aneurysm, the long duration of the headache, the persistence of paralysis between attacks, and the relatively low percentage of familial migraine in cases of this disorder have led some observers [4] to suggest that ophthalmoplegic migraine is merely a recurrent oculomotor paralysis with headache, and not a form of migraine at all.

On the other hand, one cannot escape the connection between the two syndromes. First, in large numbers of these cases an obvious migraine syndrome has been present for many years, later to be associated with recurrent oculomotor paralysis without residual paralysis between attacks.

Secondly, many migraine sufferers report that diplopia occurs regularly during attacks. While manifest paralyses are somewhat uncommon in simple migraine, this diplopia is often indicative of minimal involvement of the oculomotor nerve.

Thirdly, the clinical symptoms and signs of both ophthalmoplegic migraine and simple migraine in their most typical form have been produced by organic intracranial lesions, such as brain tumor, subdural hematoma, and intracranial aneurysm. In these cases, however, the initial transient symptoms rapidly progressed to irreversible signs; the temporary ophthalmoplegia became a permanent oculomotor paralysis, and the scintillating scotoma became a homonymous hemianopsia.

Lastly, a comprehensive review of over 1,600 cases of migraine, culled by Donahue [5] from the literature, demonstrated that various ocular paralyses occurred in 17%. In most instances the paralysis lasted for three to four days, slowly passing away but occasionally persisting for weeks or longer. In some recorded cases recovery became less perfect as the attacks persisted, and finally the paralysis became permanent (in one case in the absence of further headache). Diplopia was reported in 14% of the cases; involvement of the third nerve occurred in 80% of cases with muscle paralyses. Paralysis of ocular muscles was recorded in 296 cases, with no case of complete bilateral paralysis or bilateral ptosis. A unilateral dilated and fixed pupil was seen in 76 cases. A unilateral dilated pupil with preservation of light reaction was noted in 49 additional cases, and inequality of the pupils was recorded in 423 cases.

When the etiologic factor in the production of the symptoms of both simple and ophthalmoplegic migraine is physiologic or vasomotor, the reversibility of the process is easily understood, and the difference between the two syndromes is largely a question of severity and degree.

Classic migraine, also variously known as ophthalmic migraine, simple migraine, or migraine, has been defined by Riley [6] as "a periodic incapacitating headache culminating in nausea or vomiting, often preceded by visual disturbances, followed by sleep and occurring against the background of relatively perfect health."

While this definition may fit the picture of the "typical case," there is such variety in the symptom complex of migraine as to preclude a true clinical symptomatology. Cases of unquestioned migraine are seen in which the headache is missing from the

4. Duke-Elder, W. S.: Text-Book of Ophthalmology, Vol. 4, St. Louis, C. V. Mosby Company, 1949, p. 4131.

5. Donahue, H. C.: Migraine and Its Ocular Manifestations, Arch. Ophth. **43**:96 (Jan.) 1950.

6. Riley, H. A.: Migraine, Bull. Neurol. Inst. New York **2**:429 (Nov.) 1932.

symptom complex but in which the scintillating scotoma, homonymous hemianopsia, and nausea leave no doubt as to the diagnosis. Any single symptom or combination of symptoms and signs may appear in such a way as to establish firmly the nature of the disturbance and yet have little resemblance to the "classic migraine attack." Likewise, the degree and severity of the attack may vary within such wide limits as to be hardly noticeable, on the one hand, or to cause complete prostration of the victim, on the other. About the only thing common to most cases of migraine is the personality pattern underlying the condition—a pattern so characteristic as to be of the utmost importance in both diagnosis and treatment and one which has been ably described by Alvarez [7] and numerous other authors, including Wolff.[8]

Wolff's [8] concept of the pathogenesis of migraine is now generally accepted. He demonstrated that the preheadache phenomena of migraine (aura, irritability, scintillating scotoma, and hemianopsia) are due to vasoconstriction of cerebral arteries and that the headache is brought about by vasodilatation of vessels of the scalp and dura, primarily branches of the external carotid artery.

When dilatation of cranial arteries is prolonged over several hours, a transient thickening of their walls results, and this may have a definite effect in producing unilateral intracranial volume changes of considerable degree.

The intracranial volume changes from one phase of a migraine attack to the next have been vividly demonstrated by Goltman [9] in a patient with a defect of the left frontal bone. This patient had had migraine attacks since childhood. Immediately preceding the headache the patient's face became blanched and there was a definite depression at the site of the bone defect as compared with its usual state. As the headache developed, the depression filled until ultimately the intracranial contents protruded, assuming the appearance of a tumor. At this point the patient began to vomit and the face became flushed. With the subsidence of the headache, in 12 to 72 hours, the intracranial contents resumed their former relation to the bone defect.

Naffziger [10] was able to show these intracranial volume changes in migrainous patients in a different way. In two patients with a long history of typical migraine he demonstrated, by means of roentgenograms taken before and during the headache phase of a migraine attack, a lateral shift of the calcified pineal gland away from the side of the hemicrania.

The pathogenesis of ophthalmoplegic migraine is unknown. The prevailing theories are largely vascular in concept, but much confusion exists, and the five cases in which the clinical diagnosis of ophthalmoplegic migraine was made and autopsy was performed have shed little light on the problem. Weiss [11] found warty granulations on the left oculomotor nerve and over the base of the brain in which tubercle bacilli were found. Richter [12] described a case of a fibrochondroma involving the

7. Alvarez, W. C.: How to Live with a Migraine Headache, Cosmopolitan, p. 65, May, 1952.

8. Wolff, H. C.: Headache and Other Head Pain, New York, Oxford University Press, 1948.

9. Goltman, A. M.: The Mechanism of Migraine, J. Allergy **7**:351 (May) 1936. Wolff,[8] p. 261.

10. Naffziger, H. C.: Personal communication to the authors.

11. Weiss, E.: Ein Fall von periodisch auftretenter totaler linkseitiger Oculomotorislähmung, Wien. med. Wchnschr. **35**:521, 1885.

12. Richter, A.: Ein Fall von typisch recidivierender Oculomotorislähmung mit Sectionsbefund, Arch. Psychiat. **18**:259, 1887.

right oculomotor nerve. Karplus [13] reported a case of parenchymatous syphilis of the brain with a neurofibroma of the right oculomotor nerve. Shinoya's [14] case was one of tuberculous meningitis with enlargement of the right oculomotor nerve due to connective tissue proliferation. Alpers and Yaskin [2] observed nothing at autopsy which would account for oculomotor paralysis.

In the main, theories as to the mechanism of the production of ophthalmoplegic migraine are postulated on pressure on the oculomotor nerve from dilatation of the large vessels which are in close anatomical relation to the nerve. Bramwell [15] considered that the paralysis was due to pressure upon the nerve by a leaking aneurysm, an intact aneurysm, or an artery with an abnormal relation to the nerve. Symptoms resulted when the vessel or the aneurysmal sac was temporarily distended as a result of vasomotor changes, such as occur in an attack of migraine.

Ehlers [16] pointed to the posterior cerebral artery as the anatomical basis of a functional connection between the occipital cortex and the homolateral oculomotor nerve. The posterior cerebral artery, running in intimate relation to the third nerve, is the sole vascular supply of the visual area in the calcarine cortex, and he suggested that vascular changes in the occipital lobe are the cause of the various visual phenomena associated with simple migraine, while pressure of the same vessel on the third nerve would account for the paralysis seen in ophthalmoplegic migraine.

We have recently studied a case in which typical hemicrania and transient homonymous scintillating scotoma were followed by the development of permanent homonymous hemianopsia, which was found to be due to a leaking aneurysm of the anterior communicating artery, demonstrated by arteriography and later by intracranial exploration. The site of the lesion was far distant from the vascular supply of the calcarine fissure, and in no way related to the oculomotor nerve. There was transient, very slight dilatation of the homolateral pupil, associated with increased intracranial pressure, relieved by clipping the vessel.

Angioneurotic edema, with or without transitory hypophysial swelling, was suggested as a cause by Elliot,[17] and Fry and Schlezinger [3] recently reported a case of recurrent oculomotor paralysis associated with angioneurotic edema.

All these theories, however, are in conflict with the careful studies of Wolff [8] and his co-workers, who, as previously mentioned, have shown that migraine headache is due to distention and stretching of the branches of the external carotid artery, none of which are in close proximity to the oculomotor nerve.

For a number of years it has been well known that herniation of the hippocampal gyrus of the temporal lobe over the free edge of the tentorium, due to an ipsilateral expanding cerebral lesion, can produce pressure on the oculomotor nerve as it

13. Karplus, J. P.: Migraine ophthalmoplegique mit Autopsie, Wien. klin. Wchnschr. **8**:883, 1895.

14. Shinoya, F.: Ein Fall von recidivierender Oculomotorislähmung (Migraine ophthalmoplegique) mit Autopsie, Deutsche Ztschr. Nervenh. **42**:155, 1911.

15. Bramwell, E.: Etiology of Recurrent and Periodic Ocular Palsy and Ophthalmoplegic Migraine, Tr. Ophth. Soc. U. Kingdom **54**:214, 1934.

16. Ehlers, H.: Pathogenesis of Ophthalmoplegic Migraine, Acta psychiat. et neurol. **3**:219, 1928.

17. Elliot, A. J.: Ophthalmoplegic Migraine (with Report of a Case), Canad. M. A. J. **43**:242 (Sept.) 1940.

crosses the body of the sphenoid bone. This pressure may be sufficient to paralyze the nerve partially or completely. Herniation of the hippocampal gyrus has not heretofore been suggested as a factor in the pathogenesis of recurrent oculomotor paralysis because recurrence of the paralysis has not been a prominent feature of the herniation.

Meyer,[18] in 1920, first described temporal herniation through the incisura tentorii and pointed out that the uncus is the portion of the brain which, from its position, is most likely to herniate. Since then numerous reports have testified to the localizing value of unilateral dilatation of the pupil in cases of extradural hemorrhage, tumor, abscess, or any unilateral intracranial expanding lesion. Autopsy reports have amply demonstrated that this pupillary dilatation is produced by pressure of the herniated uncus on the homolateral third nerve.

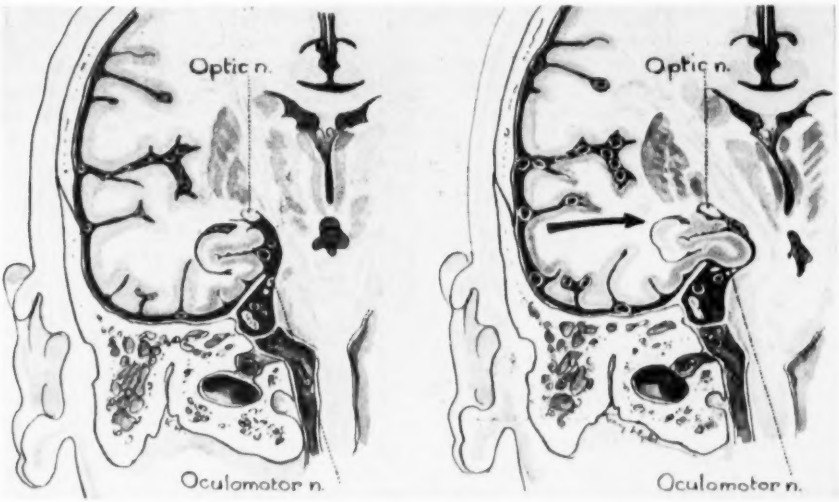

Fig. 1.—Section of the brain showing (*a*) normal relations of the hippocampal gyrus to the tentorium and the oculomotor nerve; (*b*) herniation of the hippocampal gyrus over the free edge of the tentorium, with pressure on the third nerve.

Holman and Scott [19] and Cairns [20] emphasized the occasional transient character of this pupillary reaction. Woodhall and associates [21] stated that "a dilated and fixed pupil, either of permanent or transient nature, remains the most valuable lateralizing sign and may in many instances be caused by herniation of the corresponding hippocampal gyrus."

18. Meyer, A.: Herniation of the Brain, Arch. Neurol & Psychiat. **4**:387 (Oct.) 1920.

19. Holman, E., and Scott, W. M. J.: Significance of Unilateral Dilation and Fixation of Pupil in Severe Skull Injuries, J. A. M. A. **84**:1329 (May 2) 1925.

20. Cairns, H. W. B.: Observations on the Localization of Intracranial Tumors: The Disclosure of Localizing Signs Following Decompression or Ventriculography, Arch. Surg. **18**:1936 (April, Pt. 2) 1929.

21. Woodhall, B.; Devine, J. W., and Hart, D.: Homolateral Dilation of the Pupil, Homolateral Paresis and Bilateral Muscular Rigidity in the Diagnosis of Extradural Hemorrhage, Surg., Gynec. & Obst. **74**:391 (Feb.) 1941.

The work of Reid and Cone [22] is most convincing in this regard. They have observed such herniation in cases of head injury and noted its relation to homolateral dilatation of the pupil. They have confirmed the mechanism of its production by experimental work on monkeys and proved beyond doubt the relation of unilateral increased intracranial pressure to herniation of the hippocampal gyrus and peripheral third nerve paralysis. A description of their experiments clearly demonstrates the reversibility of the process.

The extradural compression in the experimental acute herniations was obtained through the use of a Forbes window screwed tightly into a trephine hole in the skull, the position of which varied in different animals between the frontal, parietal and temporal regions. After the dura had been separated from the calvarium for a short distance around the trephine hole, Ringer's solution was allowed to flow in, and the pressure exerted could be altered and recorded at all times. . . .

It was possible to produce at will an ipsilateral pupillary dilatation and to watch the pupil return to normal on release of the pressure. After the pressure had been kept at a level

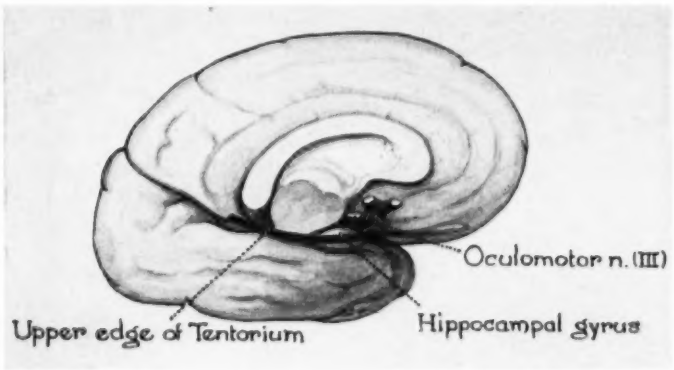

Fig. 2.—Normal relation of the upper edge of the tentorium to the uncus.

sufficiently high to hold the pupil in complete dilatation and fixation, the animals were killed. Ipsilateral hippocampal herniation was found as a routine at necropsy. [The degree of pupillary change was determined by the degree of herniation.]

Jefferson's [23] description of the anatomical relations of the tentorium is of especial interest.

No perfect illustration of the tentorium seems to exist—none that gives a correct impression of its anteroposteriorly convex upper surface, its lateral decline and the manner of the abrupt falling away of the almost vertically directed anterior 2 cm. or so of its surface, as it changes its architecture to sweep forward onto the sides of the cavernous sinus. The temporal lobes lie on the tentorium, which slopes away laterally as a gently inclined plane, so that pressure from above will tend to make them slide away from the midline. However, if one lobe is enlarged it cannot escape overhanging the free edge. For this reason, a tumor of the temporal lobe will be the surest way of bringing it more firmly into contact with the midbrain and squeezing its inner border over the sharp edge of the falx, into a situation in which it can herniate downward into the posterior fossa. . . . There is no doubt that if the tentorium were horizontal herniation would be more common than it is. Its tentlike form causes it to protect the midbrain.

22. Reid, W. L., and Cone, W. V.: Mechanism of Fixed Dilation of the Pupil, Resulting from Ipsilateral Cerebral Compression, J. A. M. A. **112**:2030 (May 20) 1939.

23. Jefferson, G.: The Tentorial Pressure Cone, Arch. Neurol. & Psychiat. **40**:857 (Nov.) 1938.

It is clear that herniation of the hippocampal gyrus is not simply the result of generalized increase in intracranial pressure, which oftener produces paralysis of the sixth nerve. Only the unilateral feature of the process can explain the localizing value of homolateral pupillary dilatation in head injury, tumor, and the like.

From clinical, pathological, anatomical, and experimental evidence it is plain that herniation of the hippocampal gyrus is due to relative increase in pressure in the ipsilateral half of the cranial cavity and that it is often a transient and reversible process. The idea that uncal herniation is catastrophic and irreversible has arisen because the condition is most frequently seen at autopsy in fatal cases with sustained high intracranial pressure.

The following case, in which autopsy revealed marked herniation of the hippocampal gyrus of the temporal lobe, producing recurrent oculomotor paralysis, suggests the pathogenesis of ophthalmoplegic migraine.

The patient had six known attacks of transient paralysis of the right third nerve, each associated with paroxysmal exacerbation of a right frontal headache, occurring over a period of about 16 days. The first attack was observed as transient diplopia by the patient himself. The second attack was observed as a temporary divergent strabismus by the patient's brother. The last four attacks were observed by us from onset to conclusion and presented the typical clinical picture of ophthalmoplegic migraine as follows:

The patient was brought to the ophthalmology department in a horizontal position on a wheeled stretcher. He was not complaining of discomfort or other symptoms, but on being questioned admitted to a slight generalized headache. He was alert, well oriented, and cooperative; was able to walk well, and did not look particularly ill. Shortly after sitting up in a chair for purposes of visual-field study, he began to complain of terrific right-sided headache and was obviously in extreme distress. From being alert and cooperative, he rapidly became very inattentive and would not respond except to loudly and sharply spoken command, and then very slowly. His face became pale, and he perspired profusely. At the same time he complained of intense urgency to urinate and, in fact, did lose urinary sphincter control. These prodromal symptoms had lasted about three minutes when it was noted that his right eye was beginning to diverge, and the patient admitted to diplopia when questioned. Within the next 30 seconds there developed complete paralysis of the right internal, superior, and inferior rectus muscles and the right inferior oblique, as well as wide dilation of the right pupil, which was fixed to light and in accommodation. Two minutes later paralysis of the levator muscle of the right lid had developed, with resulting complete ptosis. The patient was nauseated and groaning with pain and was returned to his horizontal position on the cart, where he lay on his right side. Within five minutes the hemicrania had disappeared, and the patient was again alert and cooperative, with normal skin color and absence of perspiration. Examination revealed complete recovery from the oculomotor paralysis, with normal and equal palpebral fissures, normal ocular rotations, and normal pupillary size and reactions.

Three additional, practically identical, attacks were observed. Each appeared to be precipitated by changes in the patient's position. The last attack developed into permanent ophthalmoplegia, which lasted for four days, until the patient's death, following internal carotid arteriography.

In discussing this case with Dr. Edwin Boldrey,[24] we were referred to the work of Reid and Cone [22] as a possible explanation of the oculomotor paralysis.

Autopsy revealed a bronchogenic carcinoma of the lung with metastasis to the right frontal and parietal lobes of the cerebrum. There was obvious and pronounced herniation of the right hippocampal gyrus, which had compressed and stretched the right oculomotor nerve at the incisura of the tentorium.

24. Boldrey, E.: Personal communication to the authors.

REPORT OF CASE

L. D. S., aged 50, was admitted to the neurosurgical service of the Veterans Hospital at Fort Miley, San Francisco, on Nov. 6, 1951, with the complaint of generalized headache with severe paroxysmal exacerbations associated with difficulty in concentration. He had lost 50 lb. (22.7 kg.) in weight in six months. During the preceding three weeks he had noted periodic dizziness, most marked on change of position. He had also noted periodic loss of urinary sphincter control, a peculiar taste to all his food, and marked diminution in his sense of smell. The patient had experienced diplopia on one occasion, and his brother had noted a divergent strabismus, associated with an attack of extreme head pain. The patient's family had noticed that between attacks of headache he had seemed much improved, both physically and mentally.

Physical examination showed an entirely normal condition except for the following changes: There was bilateral partial anosmia, severer on the left side. The right pupil was slightly larger than the left (subsequent examination revealed equality of the pupils), but both reacted well to light and in accommodation. Visual acuity was 20/20 in each eye. The visual fields were normal

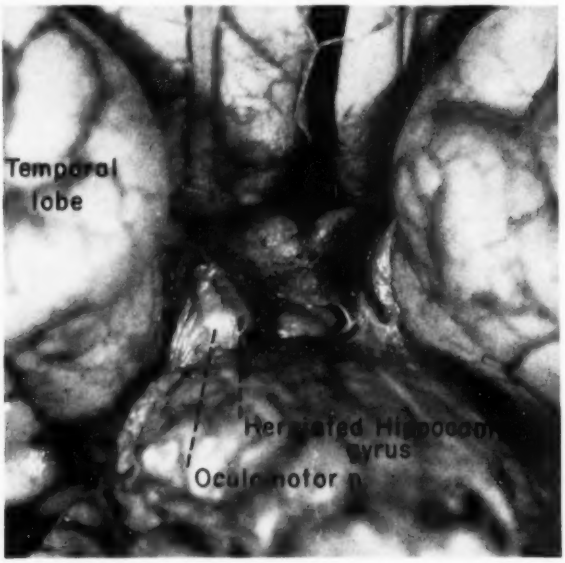

Fig. 3.—Ventral surface of the brain showing herniated hippocampal gyrus exerting pressure on the oculomotor nerve.

for multiple isopters. There was a bilateral papilledema of 2 D elevation with a few retinal hemorrhages near the disks. The tongue deviated slightly to the left, and hypalgesia of the left side of the face and weakness of the left masseter muscle were noted. Hypalgesia on the right side of an indefinite nature and an equivocal Babinski reflex bilaterally were recorded.

X-rays of the skull showed an area of irregular thinning of the inner table, sharply circumscribed in each frontal area, presumably due to enlarged diploic vessels.

Roentgenograms of the chest revealed a hemisphere of soft-tissue density in the right upper lung field, which was sharply circumscribed and suggested bronchogenic carcinoma.

An electroencephalogram was reported as diffusely abnormal with a questionable focus in the right temporal region, mostly in the anterior portion.

Lumbar puncture revealed a pressure of "over 600 mm. of water," with 40 cells per cubic millimeter, 90% of which were red blood cells.

On Nov. 6, 1951, the patient was observed in a typical attack of ophthalmoplegic migraine, which began as an extremely severe headache on the right side, followed by the gradual development of total paralysis of the right oculomotor nerve. The attack seemed to be precipitated by the movement incident to physical examination and subsided completely after a few minutes' rest, when the headache and all evidence of the third nerve paralysis disappeared. Two more attacks, identical with the first, were observed on Nov. 7, 1951. Each attack was ushered in by extreme headache on the right side, loss of attention and ability to respond to command, pallor and cold sweat, and loss of urinary sphincter control. These symptoms were followed in one or two minutes by gradual dilatation of the right pupil and complete paralysis of all the extraocular muscles supplied by the right third nerve. Finally, about six minutes from the onset of the attack, complete ptosis of the right upper lid developed. After a few minutes of rest in the horizontal position, the process reversed itself entirely. There were gradual subsidence of the headache, clearing of the mentality, and restoration of function in the intraocular and extraocular muscles.

On Nov. 8 a fourth attack of ophthalmoplegic migraine was witnessed, but this attack did not reverse itself. The headache continued, and the ophthalmoplegia remained as a permanent and complete oculomotor paralysis, until the patient's death, on Nov. 13.

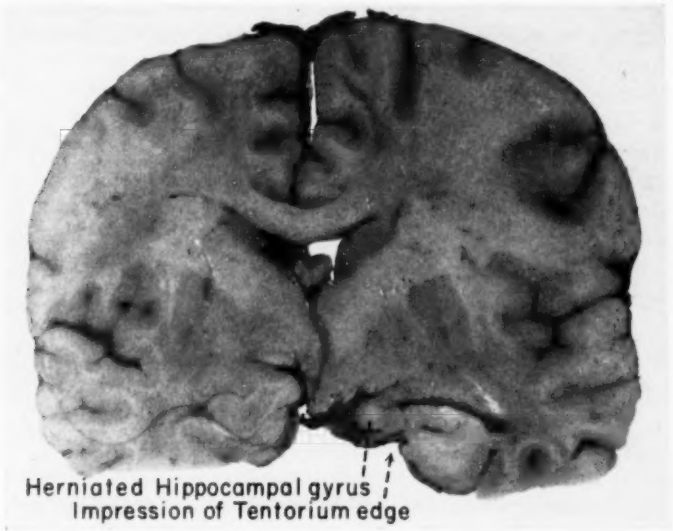

Fig. 4.—Coronal section of the brain showing herniation of the uncus with indentation of the brain by the incisura of the tentorium.

After Nov. 8 the patient grew rapidly worse and, because of the possibility that the oculomotor paralysis was due to an intracranial aneurysm, arteriography was performed on Nov. 13. The patient died that evening.

The arteriograms demonstrated a large tumor in the right frontal lobe, with a smaller lesion in the temporal lobe. The right middle and anterior cerebral arteries showed a shift to the left.

Autopsy revealed a bronchogenic carcinoma of the lung with cerebral metastasis. In the right frontal lobe, 1 cm. from the tip and 2 cm. from the midline, there was a hard round heavy, solid tumor, which shelled out completely and measured 3 cm. in diameter. There was intense edema of the brain posterior to the tumor. In the right parietal area a similar tumor was found 2 cm. from the midline and 7 cm. from the tip of the occipital lobe.

Examination of the uncut brain revealed a pronounced herniation of the right hippocampal gyrus over the edge of the tentorium with pressure on and stretching of the right third nerve. Further studies of the cut brain revealed the extent of the herniation and the degree of pressure on the oculomotor nerve. As the probable cause of death, there were many hemorrhages in the brain stem (Jefferson [23]).

COMMENT

Recurrent oculomotor paralysis with the typical clinical picture of ophthalmoplegic migraine was here produced by herniation of the hippocampal gyrus of the temporal lobe over the free edge of the tentorium, with pressure on the oculomotor nerve. The uncal herniation was secondary to unilateral cerebral edema with changes in brain volume such as to force the uncus through the incisura tentorii and onto the third nerve.

In this case the recurrent and reversible oculomotor paralysis did not occur as "a syndrome of vascular origin.[16]" It was not due to "vasomotor phenomena.[15]" It was not the result of a "leaking or intact aneurysm.[15]" But so far as any of these factors is capable of producing a unilateral increase in cerebral volume, they could have produced the same result as was produced by brain tumor in this case.

From the purely anatomical point of view, it is not difficult to explain the mechanism of production of herniation of the hippocampal gyrus and, conversely, the reversibility of the process. The anatomical relationships of the structure have

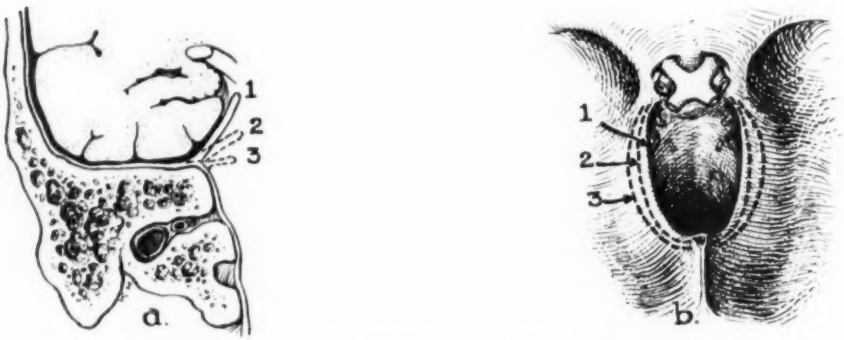

Fig. 5.—Exaggerated diagrammatic representation of possible variations in (*a*) the slope of the tentorium; (*b*) the size of the hiatus.

been succinctly described by Jefferson,[23] as previously noted. Just as variations in the degree of unilateral increase in cerebral volume would account for the occurrence of temporal herniation in some cases and not in others, so also anatomical variations in the slope of the tentorium and the size of the hiatus might account for greater ease of herniation in some persons than in others. The more horizontal the tentorium and the larger the hiatus, the less force would be required to produce the hernia of the brain. On the other hand, the anatomy of the tentorium is such that a herniation of the gyrus would strongly tend to right itself if the pressure on it were relieved, reversing the symptoms produced by the hernia.

These anatomical variations might well explain the fact that while some subdural hematomas, some temporal lobe tumors, and even some frontal lobe tumors produce uncal herniation, most do not. In some patients migraine headache is repeatedly followed by oculomotor paralysis, whereas in most cases ophthalmoplegia never occurs.

In this regard the most recent case of Alpers and Yaskin [2] is of interest to us.

The patient exhibited typical clinical ophthalmoplegic migraine with recurrent paralysis of the left oculomotor nerve and headache. Intracranial aneurysm was suspected, and arteriography of the left internal carotid artery and exploratory craniotomy were performed, after which the patient became comatose and died. Autopsy revealed thrombosis of the internal carotid artery, an extradural hematoma, herniation beneath the falx of the medial surface of the left hemisphere, and herniation of the left hippocampal gyrus. No aneurysm or vascular anomaly was found to account for the ophthalmoplegic migraine. Even if we believe that in this case the herniation was secondary to the unilateral cerebral edema produced by the thrombosis of the internal carotid artery and the subdural hematoma, it is interesting to note that here was a patient in whom temporal herniation was likely to occur and that there is no other explanation for the preoperative existence of typical ophthalmoplegic migraine of many years' duration.

The role of uncal herniation, with or without ophthalmoplegia, is important in explaining certain other phenomena associated with migraine.

When one considers that the ocular muscle paralyses reported by Donahue,[5] and noted above, were in cases of ordinary migraine, and not of ophthalmoplegic migraine, the very high incidence of involvement of the ocular musculature (especially that under control of the oculomotor nerve) is indeed striking and indicates a much greater frequency of this phenomenon in simple migraine than has hitherto been suspected. No other mechanism would explain these findings as well as does the fact of herniation of the temporal lobe of varying degree.

The occurrence of contralateral transient hemiplegia and paresthesias of varying degree in association with attacks of simple or classic migraine is well known and can best be explained by compression of the crus and pyramidal tracts by herniation of the brain through the incisura tentorii.

The pain of ophthalmoplegic migraine is generally described as quite different from ordinary migraine. It is severer, usually of longer duration, often precedes or coincides with ocular paralysis, is definitely localizable, but trigeminal in distribution, and could easily be accounted for by the complications of herniation of the hippocampal gyrus. It is probably due to the pressure of the herniated uncus on the branches of the trigeminal nerve, which richly supply the area below it.

Visual defects are frequently associated with both classic and ophthalmoplegic migraine. In the former the so-called scintillating scotoma is the predominant symptom, occurring in a high percentage of cases and often merging into transient homonymous hemianopsia, which is succeeded by hemicrania.

For the most part, the visual defects are transient, and even evanescent. The scintillating scotomas are usually clearly homonymous and are often described as crude, half-formed, and bizarre visual hallucinations. The homonymous hemianopsia is usually very complete, but not dense in character, and lasts 20 or 30 minutes. It is Wolff's[8] view that the preheadache visual phenomena of migraine are due to cranial vasoconstriction and are localizable in the visual cortex in the occipital lobe. They result from occlusion of the branches of the posterior cerebral arteries, causing ischemia of the most peripheral portions of the blood supply of the artery, that area near the occipital pole, and spread ultimately to involve the anterior portions of the calcarine fissure and hence the peripheral portion of the visual field. Wolff con-

cludes, as previously noted, that the preheadache visual phenomena result from dysfunction of cerebral vessels, in contrast to the headache phenomena, which result chiefly from dilatation of extracerebral vessels.

Besides Wolff's experiments, there is considerable evidence for this hypothesis in the case reports of Adie [25] and the electroencephalographic studies of Engel and his co-workers.[26] These observers found that electrical disturbances, similar to those seen during cerebral ischemia, could be obtained from the electrodes over the occipital cortex during the scotomatous phase of migraine, both spontaneous and induced by decompression sickness.

In the first description of herniation of the hippocampal gyrus, by Meyer,[18] he described the strangulation of the posterior cerebral artery by the edge of the tentorium and the "collapse of the corresponding part of the occipito-temporal cortex." He seemed to be particularly concerned with the possibility of hemianopsia as a false localizing sign resulting from obliteration of the posterior cerebral artery. Greenfield [27] and Moore and Stern [28] were also concerned with the obliteration of the blood supply to the area striata in cases of herniation of the brain into the hiatus of the tentorium. Reid and Cone [22] reported the finding at autopsy of infarction of the ipsilateral occipital lobe in three of six cases of herniation of the hippocampal gyrus with dilated pupil.

In his description of the tentorium, Jefferson [23] dwelt specifically on this point, as follows:

> Sloping obliquely backward on either side and partly encircling the midbrain lie the posterior cerebral arteries, and it is a matter for surprise that they do not more often suffer from their close relationship to the sharp edge of the tentorium. No other intracranial vessel is vulnerable in this manner in any thing approaching the same degree.

It would appear from the above that the visual disturbances associated with ophthalmoplegic migraine may be explained on the basis of ischemia or even infarction of the occipital lobe produced by compression of the posterior cerebral artery between the sharp edge of the tentorium and the herniated hippocampal gyrus.

The visual phenomena of migraine are more transient because the physiological, vasomotor disturbances which produce the arterial dysfunction are more fleeting; even here, however, permanent damage may occur.[25]

The loss of visual field in ophthalmoplegic migraine also usually starts as a transient and reversible sign but may become permanent, just as the initially reversible oculomotor paralysis is finally made fixed.

From the observations of Naffziger,[19] Goltman,[9] and Wolff,[5] the changes in intracranial volume occurring in migraine are known. These changes, physiological, or vasomotor in origin, we consider capable of producing, in susceptible persons, a herniation of the hippocampal gyrus of a degree sufficient to compress the oculo-

25. Adie, W. J.: Permanent Hemianopia in Migraine and Subarachnoid Hemorrhage, Lancet **2**:237 (Aug. 2) 1930.

26. Engel, G. L.; Webb, J. P.; Ferris, E. B.; Romano, J.; Ryder, H., and Blankenhorn, M. A.: A Migraine-Like Syndrome Complicating Decompression Sickness: Scintillating Scotomas, Focal Neurologic Signs and Headache; Clinical and Electroencephalographic Observations, War Med. **5**:304 (May) 1944. Engel, G. L.; Ferris, E. B., and Romano, J.: Focal Electroencephalographic Changes During the Scotomas of Migraine, Am. J. M. Sc. **209**:650 (May) 1945.

27. Greenfield, J. G., cited by Jefferson.[23]

28. Moore, M., and Stern, K.: Vascular Lesions in the Brain Stem and Occipital Lobe Occurring in Association with Brain Tumors, Brain **61**:70 (March) 1938.

motor nerve and related structures. The transient character and reversibility of the symptoms are correlated with their etiology and are in direct proportion to the degree, duration, and cause of the unilateral increase in cerebral volume producing them. Thus, a phenomenon of vasomotor origin would be expected to recede with the subsidence of the "vegetative storm" which brought it about. With repeated and prolonged "attacks," certain relatively irreversible changes in the insulted tissue would eventuate, so that each new change would be added to the residuum of its predecessor until a permanent loss of function resulted.

When the cerebral edema causative of the uncal herniation is due to an organic pathological process, such as tumor or hemorrhage, the resulting clinical picture is apt to be more violent in its manifestations, with transient characteristics absent or quickly merging into permanent change. But regardless of the etiology of the unilateral increase in cerebral volume, it is the herniation of the temporal lobe that is responsible for the clinical picture of ophthalmoplegic migraine in one or another of its protean forms.

SUMMARY

1. Charcot's original definition of ophthalmoplegic migraine as "migraine associated with palsy of an ocular nerve" is still valid. The two clinical entities may be strikingly similar, and classic migraine frequently merges into the ophthalmoplegic type.

2. The various theories as to the pathogenesis of ophthalmoplegic migraine are, to date, largely pathological in concept. They have little in common with accepted ideas on the etiology of migraine.

3. Anatomical, clinical, pathological, and experimental evidence has been cited to prove that a unilateral increase in cerebral volume can produce ipsilateral herniation of the hippocampal gyrus of the temporal lobe through the incisura tentorii, resulting in recurrent homolateral oculomotor paralysis, visual disturbances, and hemicrania.

A case of recurrent oculomotor paralysis presenting the typical clinical symptom complex of ophthalmoplegic migraine is reported. Autopsy revealed a marked herniation of the hippocampal gyrus with pressure on the third nerve. The hernia was secondary to unilateral cerebral edema produced by a brain tumor.

A new theory of the pathogenesis of ophthalmoplegic migraine is advanced. It is suggested that the symptom complex is produced by herniation of the hippocampal gyrus in susceptible persons, that the herniation of the brain over the free edge of the tentorium is dependent on anatomical variations correlated with unilateral cerebral edema, and that the edema may be the result of changes of intracranial volume of physiological or pathological character.

FUNCTIONAL EVALUATION OF THE PUPILLARY LIGHT REFLEX PATHWAYS

Experimental Pupillographic Studies in Cats

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SEAN B. MURPHY, M.D.
AND
IRENE E. LOEWENFELD
NEW YORK

THE PATHWAYS of the pupillary reflex to light have been the subject of many investigations. A survey of the older literature was given by Behr [1] and one of the more recent work, by Magoun and Ranson.[2] Research included experiments by electrical stimulation and degeneration studies after lesions, in many species, especially the rat, rabbit, dog, cat, and monkey. A functional evaluation of the effect of lesions at various sites within the reflex arc was attempted.[3]

The general course of the reflex arc is now agreed upon. In cats, a majority of the retinal fibers cross in the optic chiasm, and a minority remain uncrossed (Fig. 1). The fibers run in the optic tract and pass to the pretectal area through the brachium of the superior colliculus. The existence of a synapse in this region is established, although its exact site is debated. Magoun and Ranson believed that it was in the pretectal area,[4] while Szentágothai [5] located it in the lateral geniculate body.[6] In either case a majority of the fibers of the second neuron cross from the pretectal area through the posterior commissure to the small-celled third-nerve (Westphal-Edinger) nucleus of the opposite side, while a minority of fibers arch

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1. Behr, C.: Die Lehre von den Pupillenbewegungen, Berlin, Springer-Verlag, 1924.

2. Magoun, H. W., and Ranson, S. W.: Afferent Path of the Light Reflex: A Review of the Literature, Arch. Ophth. **13**:862-874, 1935.

3. Adler, F. H.: Pupilloscopic Findings in Lesions in Different Parts of the Reflex Arc, Arch. Ophth. **55**:262-272, 1926.

4. Magoun and Ranson.[2] Ranson, S. W., and Magoun, H. W.: Central Path of the Pupilloconstrictor Reflex in Response to Light, Arch. Neurol. & Psychiat. **30**:1193-1204, 1933. Magoun, H. W.; Ranson, S. W., and Mayer, L. L.: Pupillary Light Reflex After Lesions of the Posterior Commissure in the Cat, Am. J. Ophth. **18**:624-630, 1935. Magoun, H. W., and Ranson, S. W.: Central Path of the Light Reflex: A Study of the Effect of Lesions, Arch. Ophth. **13**:791-811, 1935.

5. Szentágothai, J. L.: Die zentrale Lietungsbahn des Lichtreflexes der Pupillen, Arch. Psychiat. **115**:136-156, 1942.

6. Magoun and Ranson's opinion is based on Marchi degeneration studies by Barris, Ingram, and Ranson (Barris, R. W.: Ingram, W. R., and Ranson, S. W.: Optic Connections of the Diencephalon and Midbrain of the Cat, J. Comp. Neurol. **62**:117-153, 1935), while Szentágothai used Bielschowsky's fiber degeneration stain, modified after Gross and Reumont.

around the central gray matter to reach the constrictor nucleus on the same side. Magoun and Ranson assumed an additional, smaller decussation ventral to the Sylvian aqueduct and the central gray matter, since they found not only strong homolateral, but also weak contralateral, constriction on electrical stimulation of the fibers emerging from the posterior commissure.

The efferent pupillary constrictor path from the third-nerve nucleus to the ciliary ganglion is uncrossed. After a final synapse in the ciliary ganglion, the

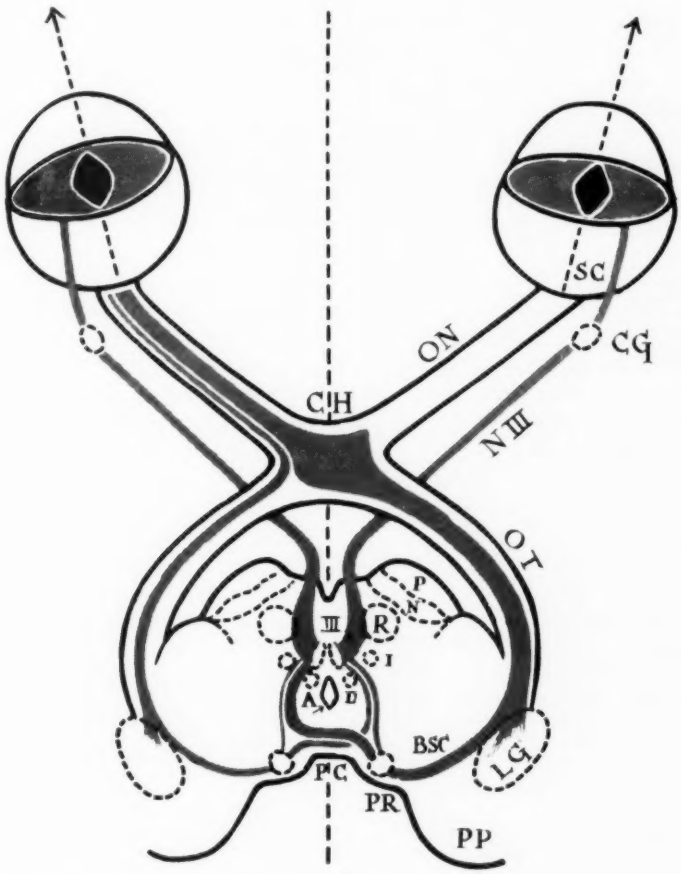

Fig. 1.—Pupillary light reflex pathways in the cat.

In contrast to the situation in primates, there is an unequal distribution of fibers, both in the optic chiasm and in the central decussation. The majority of fibers cross; the minority remain uncrossed.

A indicates Sylvian aqueduct; *BSC*, brachium of superior colliculus; *CG*, ciliary ganglion; *CH*, optic chiasm; *D*, nucleus of Darkschewitsch; *I*, interstitial nucleus of Cajal; *LG*, lateral geniculate body; *N*, substantia nigra; *NIII*, oculomotor nerve; *III*, nucleus of third nerve; *ON*, optic nerve; *OT*, optic tract; *P*, cerebral peduncles; *PC*, posterior commissure; *PP*, posterior portion of pulvinar; *PR*, pretectal region; *R*, nucleus ruber; *SC*, short ciliary nerves.

impulses reach the iris constrictor via the short ciliary nerves. In cats, only a few, if any, postganglionic sympathetic fibers from the superior cervical ganglion pass through the ciliary ganglion, as they do in man and monkeys.

Although the general course of the reflex arc is, therefore, fairly well defined, earlier attempts at evaluation of the functional effect of lesions at different sites within the reflex arc were hampered by the difficulties of observation with the unaided eye. Such observations are necessarily subject to errors for it is not possible to judge the exact relationship of the simultaneous reactions of the two pupils or to detect reliability and evaluate any but gross abnormalities in their motility.

It is the purpose of the present study to determine, chiefly with a clinical goal in mind, the functional value of the neurons which constitute the pathway of the pupillary reflex to light. For the purposes of this study, the intraretinal neurons are disregarded, and the term "first neuron" is applied to the ganglion cell layer, which sends fibers through the optic nerve and tract. The following questions will be considered:

1. What is the comparative pupillomotor value of the fibers which cross and those which do not cross in the optic chiasm of the cat? What functional defects result from section of (*a*) one optic nerve and (*b*) one optic tract respectively in the reactions of the homolateral and contralateral iris?

2. How are the impulses which reach the pretectal area distributed in the second neuron, which extends from the pretectal area of either side to the homolateral and the contralateral third-nerve nucleus? Is the central distribution of impulses an independent function of the second neuron, or does it depend on the distribution of impulses in the first neuron? Clinically speaking, can anisocoria be induced by damage to the first neuron?

3. Is it possible to distinguish between lesions in the afferent part of the light reflex pathways and those in the third-nerve nucleus, the efferent third-nerve fibers, or the ciliary ganglion?

METHOD

Eighteen cats were used in the present study. The conclusions of this investigation are also based on data from additional cats and monkeys in which lesions were made during the course of earlier experiments.

Section of the right optic tract was performed in nine cats. The right side of the skull was opened by trephination of the frontoparietal bone, and the opening was enlarged with rongeurs. The dura was opened and the brain deflected medially by means of a spatula. Thus, the right optic nerve, the optic chiasm, and the right optic tract could be seen. The right optic tract was sectioned with the point of a Graefe knife; with this method, hemorrhage and trauma to the surrounding structures were kept to a minimum.

In three of the nine animals the left optic nerve was cut in a subsequent operation. The technique was the same as that described above except that the opening through the skull was smaller because the optic nerve is more accessible to section. In three animals the right optic nerve and tract were cut in the same operation. Two of the animals died a week later, before pupillographic records were taken, and the results of these two experiments were based on observations with the unaided eye only.

Lesions in the region of the pretectal area and posterior commissure were produced with the aid of the Horsley-Clarke stereotaxic instrument.[6a] Electrical stimulation with a single 0.5 mm. insulated silver electrode (indifferent electrode attached to the frame of the instrument) was used to locate the region of the pretectal area or the posterior commissure. In agreement with

6a. Dr. F. A. Mettler placed the facilities of his laboratory at our disposal for these operations and aided with his advice.

Ranson and Magoun's findings, intense constriction of both pupils was noted. Fulgurating lesions were then produced at these points, a direct current of 5 ma. intensity being applied for 20 seconds.

In seven normal cats small lesions were produced in the pretectal area. In six other cats larger lesions were produced at the same site. Two of these six cats were otherwise normal. The remaining four belonged to the aforementioned group with optic-path lesions: Two of them had previous lesions in the right optic tract and the left optic nerve; one of them, in the right optic tract and nerve, and one, in the right optic tract only. In all animals, the nictitating membrane was removed from both eyes to facilitate pupillographic tests.

All cats were examined pupillographically before the operation and after a postoperative period of at least two weeks, when the acute effects of the operations had disappeared. Pupillography was done by the method and apparatus previously described.[7] After adaptation to dark red-infrared illumination, records were taken while the right or the left eye was exposed to series of light stimuli. In order that emotional excitement should be prevented from inhibiting the contractions to light, the animals were wrapped in towels and elastic bandages, in which they rested comfortably. Cats which were difficult to handle were not used.

After the animals were killed, the extent of injury to the brain was determined histologically. Serial sections were made through the optic chiasm and tracts and through the midbrain, and selected slides were prepared by the Weigert-Pal stain for myelin, the Davenport silver stain, or the hematoxylin-eosin stain.

RESULTS

1. *Pupillogram of a Normal Cat* (Fig. 2).—The reflex to light in normal cats was described in an earlier paper.[7b] When the cat is calm, the horizontal diameter of the dark-adapted pupil ranges from 8 to 12 mm. When a light stimulus of medium, 15 foot-candle (15 f.-c.) intensity and one-second duration is applied to one eye, bilateral contraction takes place, after a latent period of about 0.2 second. The extent of contraction varies in different animals from 4 to 6 mm.; the duration, from 1.2 to 1.6 seconds. Contraction is followed by redilation, which is slower and less extensive than the preceding contraction, so that in normal, nonexcited cats the pupil does not recover its dark-adapted diameter but becomes smaller and smaller as the number of light reflexes increases.

In normal cats, the direct reaction to light is often more extensive than the indirect one, resulting at the peak of contraction in slight anisocoria (Fig. 2). The development of such a dynamic contraction anisocoria—definitely pathological in man and monkeys—is normal in cats and is explained by the uneven distribution of crossed and uncrossed fibers in the optic chiasm and central decussation. When, for example, the right eye is stimulated by light, the majority of impulses cross through the optic chiasm to the left pretectal area. From there the majority of impulses cross back to the right third-nerve nucleus. This causes the right pupil to contract more extensively than the left one. In accordance with the rule that nerve centers fatigue faster on stimulation with a weak than with a strong stimulus,[8] the left third-nerve nucleus tires sooner than the right one. Thus, the already less-extensive reactions of the left pupil diminish faster than those of the right, and anisocoria increases on repeated stimulation.

7. Lowenstein, O., and Loewenfeld, I.: (a) Role of Sympathetic and Parasympathetic Systems in Reflex Dilatation of the Pupil: Pupillographic Studies, Arch. Neurol. & Psychiat. **64**:313-340, 1950; (*b*) Mutual Role of Sympathetic and Parasympathetic in Shaping of the Pupillary Reflex to Light: Pupillographic Studies, ibid. **64**:341-377, 1950.

8. Lowenstein, O., and Loewenfeld, I.: Disintegration of Central Autonomic Regulation During Fatigue and Its Reintegration by Psychosensory Controlling Mechanisms: I. Disintegration; Pupillographic Studies, J. Nerv. & Ment. Dis. **115**:1-21, 1952.

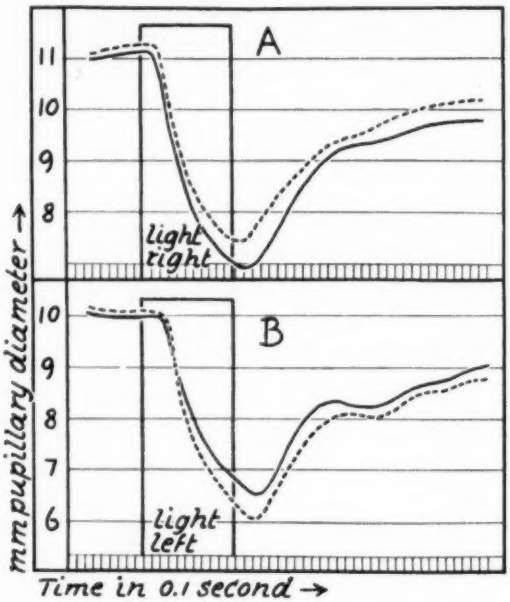

Fig. 2.—Light reflex in the normal cat. The solid line represents the right pupil; the broken line, the left pupil.

In darkness the pupils are equal. During contraction to light the direct reaction of the pupil on the side of the stimulus is slightly more extensive than the consensual reflex of the opposite pupil.

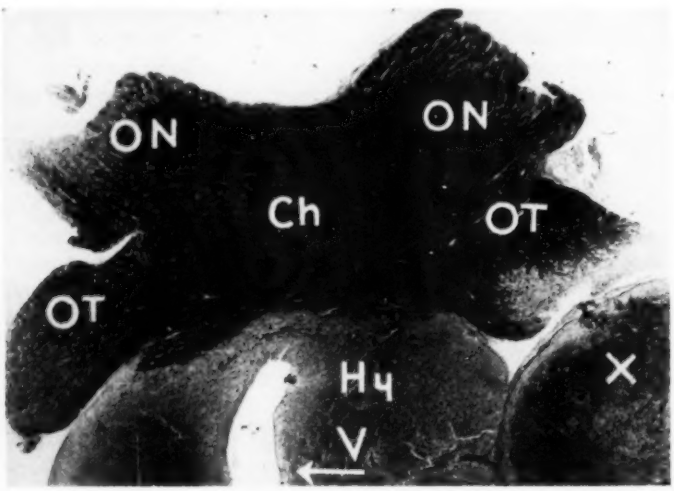

Fig. 3.—Optic chiasm of a cat in which the right optic tract had been cut.

Ch indicates optic chiasm; *Hy*, hypothalamus; *ON*, optic nerve; *OT*, optic tract; *V*, third ventricle, and *X*, necrotic area at site of lesion.

2. *Cats After Section of the Right Optic Tract.*—After section of the right optic tract (Fig. 3), the right pretectal area can no longer receive stimulation. All impulses from the right and the left retinae are mediated to the left pretectal region. From there they are unequally distributed, with a majority of impulses running to the right third-nerve nucleus and a minority to the left one. This explains our findings, which are summarized as follows:

(*a*) In darkness the pupils are equal in size (Fig. 4 *A1*), for unequal distribution of impulses is impossible in the absence of stimulation.

(*b*) When both eyes are illuminated by diffuse light, the pupil on the side of the lesion becomes smaller than its fellow pupil; this is the expression of the fact

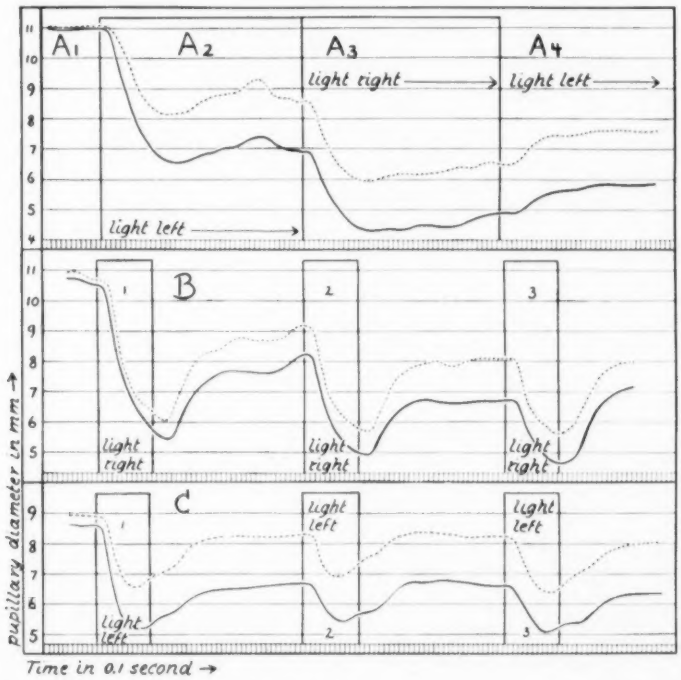

Fig. 4.—Pupillogram of a cat after section of the right optic tract. The right pupil is represented by a solid line; the left pupil, by a broken line.

A1: In darkness the pupils are equal. *A2:* When the left eye (with uncrossed optic fibers only) is stimulated, both pupils contract. *A3:* When the stimulating light beam is shifted from the left eye to the right eye, the pupils contract further. *A4:* The pupils redilate when the light is shifted back to the left eye. The right pupil contracts more extensively, regardless of which eye is illuminated.

B: On stimulation of the right eye, the pupils react normally.

C: On stimulation of the left eye, the reflexes are inextensive and show W-shapes, which are found in normal animals only upon low-intensity light stimulation; the right pupil contracts more extensively than the left, although the stimulus arises from the left retina.

that the major part of the stimuli which reach the pretectal area opposite the lesion are mediated to the third-nerve nucleus on the side of the lesion.

(*c*) When a focused beam of light is directed onto the left eye, both pupils contract (Fig. 4 *A2*), the right one more than the left (see below). When the same beam of light is suddenly shifted from the left eye to the right eye, the pupils respond with further contraction (Fig. 4*A3*). It could be objected that this might be due to the coming into play of new, previously unstimulated retinal elements, and not to a higher pupillomotor value of the fibers which arise in the right retina and cross in the optic chiasm. This assumption is disproved by the fact that both pupils dilate when, after some time, the beam of light is shifted back to the left eye (Fig. 4 *A4*). The pupillomotor forces mobilized by the crossed optic fibers greatly exceed those of the noncrossed fibers.

(*d*) When the right eye, with only crossing optic fibers remaining, is stimulated by light stimuli of standard intensity, the resulting reflexes are normal in shape and not measureably diminished; when, on the other hand, the left eye, with only uncrossed fibers remaining, is stimulated by the same light stimuli, the reactions are reduced; they show the same reflex shapes as those normally found in response to low-intensity light stimuli[9] (Fig. 4*B* and *C*).

When light reflexes are elicited, dynamic contraction anisocoria develops, whereby the pupil on the side of the lesion always contracts more extensively and fatigues less readily than the one on the other side, irrespective of which eye is stimulated (Fig. 4 *B* and *C*). The second neuron depends on fibers anterior to it for the amount of stimulation it receives. However, the distribution of these impulses to the Westphal-Edinger nucleus of either side is exclusively the function of the second neuron.

Clinical conclusions may be drawn from these facts. For example, when a syndrome, as shown in Figure 4, is found in man—where the distribution of fibers in the second neuron is about equal—it cannot be explained on the basis of a single lesion in the optic nerve or tract. An additional lesion in the second neuron must be present by which the normally equal distribution of fibers is disturbed. Clinical reports often explain anisocoria found in cases of lesions of the optic nerve or tract on the basis of unequal mediation of light stimuli in the afferent first neuron. This explanation is in conflict with the experimental facts presented in this study. The close proximity of the third nerve to the optic tract (Fig. 5) may frequently account for a mydriatic pupil with impaired light reflex on the side of an optic tract lesion.

3. *Section of the Right Optic Tract and Left Optic Nerve.*—When the left optic nerve was cut in cats in which the right optic tract had previously been severed, only crossed fibers from the right retina remained in the left optic tract (Fig. 5). Reactions of the right eye to light stimulation continued to be normal. No reaction, of course, was obtained by light stimulation of the left eye.

Attempts were made to find differences in reactivity of the nasal and the temporal half of the right retina. For this purpose, series of reflexes were elicited by a small but bright beam of parallel white light (0.75 mm. diameter), which was directed toward the temporal or the nasal half of the retina from a distance of 3 cm. and at an angle of about 30 degrees. No significant difference was found in the response of the two halves of the retina.

9. Lowenstein, O., and Loewenfeld, I.: Types of Central Autonomic Innervation and Fatigue: Pupillographic Studies, A. M. A. Arch. Neurol. & Psychiat. **66**:580-599, 1951; footnote 8.

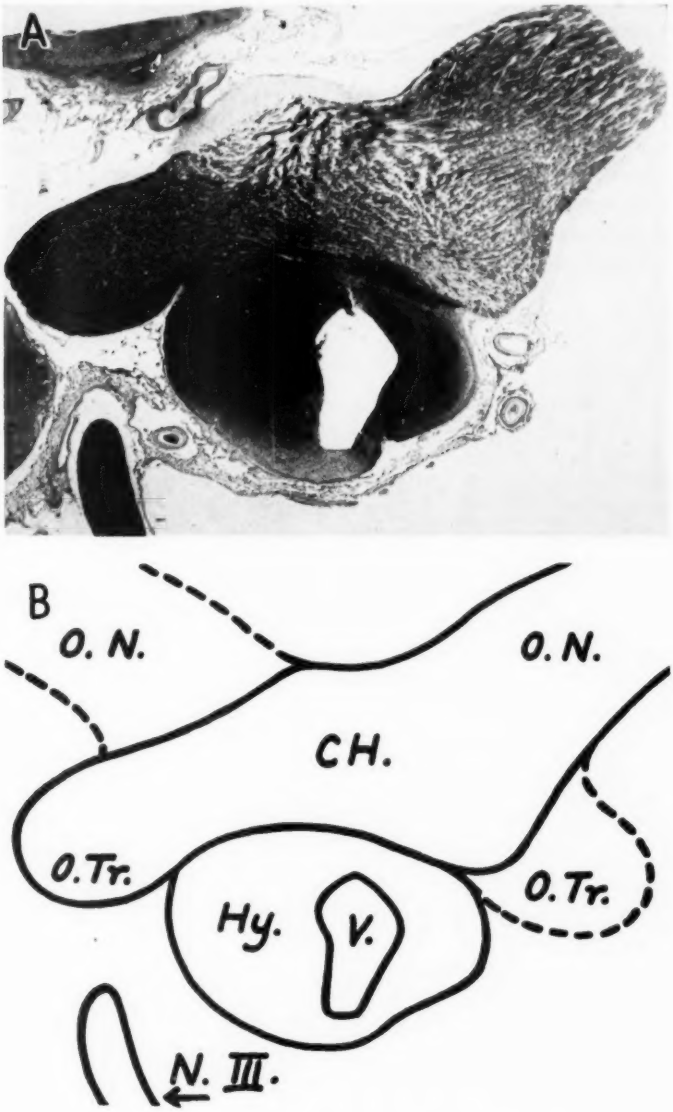

Fig. 5.—*A:* Section of optic chiasm of a cat in which the right optic tract and left optic nerve had been cut four months earlier.

B: Outline drawing of the section shown in *A*.

The solid line represents surviving structures; the broken line, degenerated left optic nerve and right optic tract.

CH. indicates optic chiasm; *Hy.*, hypothalamus; *N.III* oculomotor nerve; *O.N.*, optic nerve; *O.Tr.*, optic tract; *V.*, third ventricle.

4. *Section of the Right Optic Nerve and Tract.*—When the right optic nerve and tract were cut, only the uncrossed fibers of the left retina remained unimpaired. Vision of the animals was very poor,[10] and the pupils were large even in bright light. Reactions to light stimulation of the left eye were inextensive and showed low-intensity reflex shapes (Fig. 6 *A*). The consensually reacting right pupil contracted more extensively than the directly reacting left pupil. This preponderance of reflex activity of the right iris is understandable, since only the left pretectal area received impulses, the major portion of which were mediated to the third-nerve nucleus of the opposite side. These findings confirm that distribution of impulses to the third-nerve nuclei is a function of the central decussation alone.

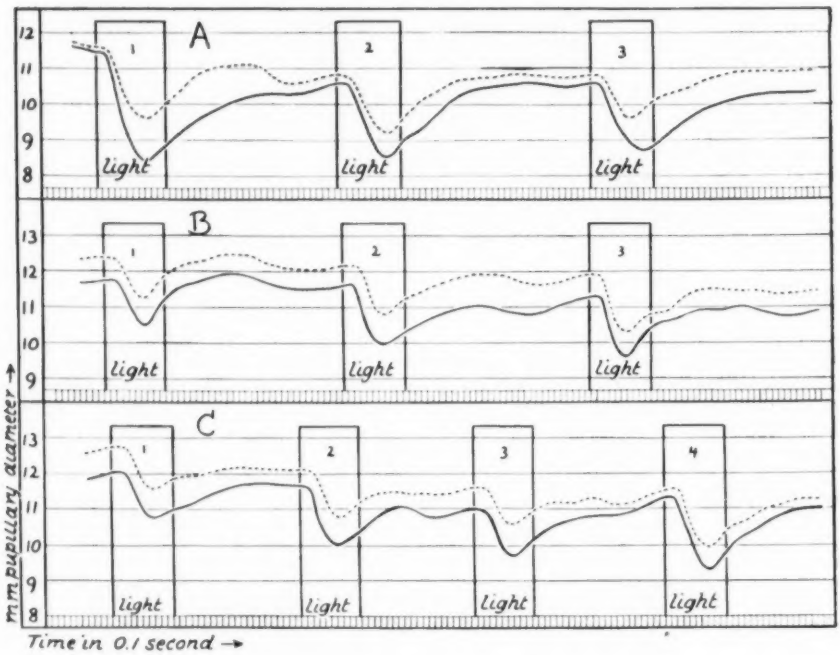

Fig. 6.—Pupillogram of a cat in which the right optic nerve and tract were cut.

The solid line represents the right pupil; the broken line, the left pupil.

A: Light reflexes elicited from the left eye by a large, centrally directed beam of light of 15 f.-c. intensity.

B: Light reflexes elicited by a bright, but small (0.75 mm. diameter) beam of parallel light directed toward the nasal half of the left retina from a distance of 3 cm. and at an approximate angle of 30 degrees.

C: Light reflexes elicited by the same light beam directed toward the temporal half of the left retina.

As in the experiments described in Section 3, series of reflexes were elicited by a small beam of parallel light; the reactions obtained were equally extensive

10. Vision was tested by observation of the general behavior of the animals and by elicitation of the optokinetic nystagmus.

whether the beam was directed toward the nasal half, the temporal half, or the center of the left retina (Fig. 6 *B* and *C*).

According to Brouwer, Zeeman, and Overbosch, as reported by B. Brouwer,[11] optic fibers which take their origin from the nasal half of the retina cross in the optic chiasm, while only part of those from the temporal half of the retina cross; i. e., each optic tract of the cat carries pupillomotor fibers from a part of the temporal half and the entire nasal half of the opposite retina.

When, in the experiments described above, only a portion of either the temporal or the nasal half of the retina was stimulated by a small (0.75 mm. diameter) beam of bright parallel light, series of bilateral low-intensity light reactions were obtained. In normal cats, and in cats whose optic tract was cut on the same or on the contralateral side, similar series of reflexes were obtained when the temporal, the nasal, or the central areas of the retina were stimulated. It is possible that dispersion of light from a bright source and the high sensitivity of the pupilloreceptor mechanism account for these results. In man, perimetric examination of pupillary reflexes with threshold-intensity light stimuli was reported by Harms.[12] If such an examination were combined with pupillography, a high degree of accuracy might be expected. The experiments with a small beam of bright light described in the present study, however, do not allow one to draw conclusions on the distribution of pupillomotor receptors in the retina of cats.

5. *Lesions in the Pretectal Region and Posterior Commissure.*—Small lesions in this area resulted in reduction of the reflexes, but not in their extinction. When a larger lesion was produced in the midline, neither pupil reacted to light, but vision seemed unimpaired. Figure 7 shows a cross section of the midbrain just anterior to the superior colliculi. The lesion, situated exactly in the midline, resulted in pupillary, but no other symptoms. As shown in Figure 8 *B*, the direct and indirect reactions to light, when either eye was stimulated, were almost completely abolished. The pupils were mydriatic (diameter, 13 mm.) even in bright light. Dilation to psychosensory stimuli was not abolished, although much reduced in extent, owing to the mechanical limit of the already mydriatic pupil. Magoun and Ranson described extreme miosis, preceding the stage of mydriasis, during the early postoperative period. This phenomenon was not observed in the present study, possibly owing to the fact that our lesions were considerably smaller than those described by Ranson and Magoun and did not extend to the thalamus.

Figure 9 shows a cross section of the midbrain of a cat at the level of the posterior commissure; the right optic tract had previously been severed (Fig. 3). A small, strictly unilateral lesion in the left pretectal area resulted in almost complete abolition of the direct and indirect reflexes to light from either eye. Figure 10 shows the pupillogram while the animal was normal (Fig. 10 *A*), after the right optic tract was cut (Fig. 10 *B*), and after unilateral lesion of the left pretectal region (Fig. 10 *C*). The already mydriatic pupils in this animal, as in the cat whose reactions are shown in Figure 8, dilated further in response to sensory stimuli.

11. Brouwer, B.: Anatomical Phylogenetical and Clinical Studies on the Central Nervous System: The Herter Lectures of the Johns Hopkins University, Vol. 17, 1926, Baltimore, Williams & Wilkins Company, 1927.

12. Harms, H.: Grundlagen, Methodik und Bedeutung der Pupillenperimetrie für die Physiologie und Pathologie des Sehorgans, von Graefes Arch. Ophth. **149**:1-68, 1949.

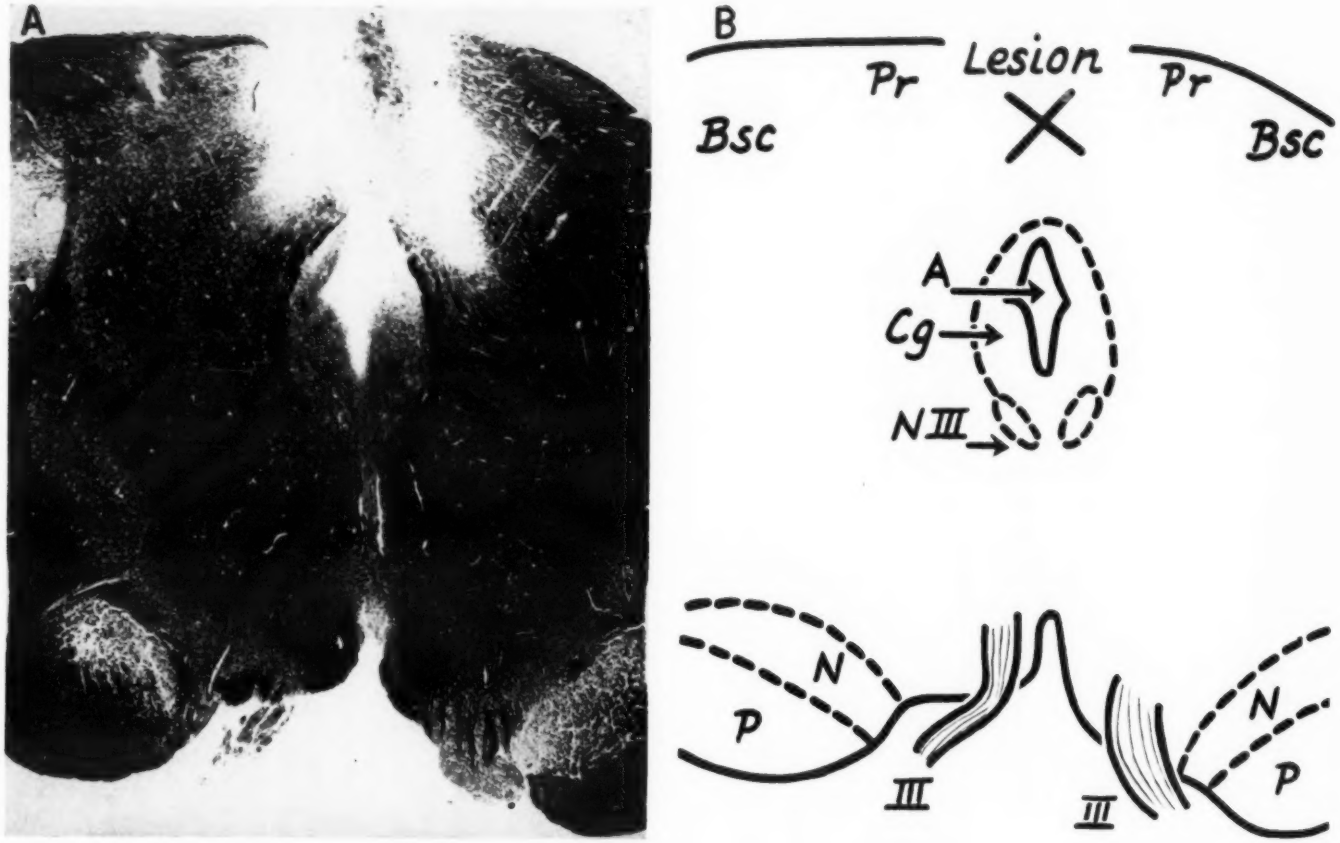

Fig. 7.—Cross section of midbrain of a cat with a large midline lesion in the region of the pretectal area.

A: Microscopic section (Davenport silver stain).

B: Outline drawing of the section shown in *A*.

A indicates aqueduct of Sylvius; *Bsc*, brachium of superior colliculus; *Cg*, central gray matter; *N*, substantia nigra; *NIII*, nucleus of oculomotor nerve; *III*, third-nerve roots; *P*, cerebral peduncles; *Pr*, pretectal region.

6. *Lesions of the Efferent Pathway.*—Pupillographic studies on the functional effect of lesions in the third-nerve nucleus, efferent third-nerve fibers, and ciliary ganglion in cats and monkeys and in clinical cases in man were described previously.[7b] The results may be summarized as follows:

(*a*) Lesions of the third-nerve nucleus may be irritative or destructive. If destruction of the nucleus is complete, the pupil on the side of the lesion becomes mydriatic and does not contract to light stimulation. If destruction of the nucleus is incomplete, the reaction to light is decreased both in extent and in speed, according to the size of the lesion. In contrast to lesions in the afferent path of the reflex arc, lesions in the efferent portion are purely unilateral, with the injured side always reacting less, both directly and consensually, while the reflexes of the contralateral pupil are normal. Psychosensory dilation is always present. In cases of irritative

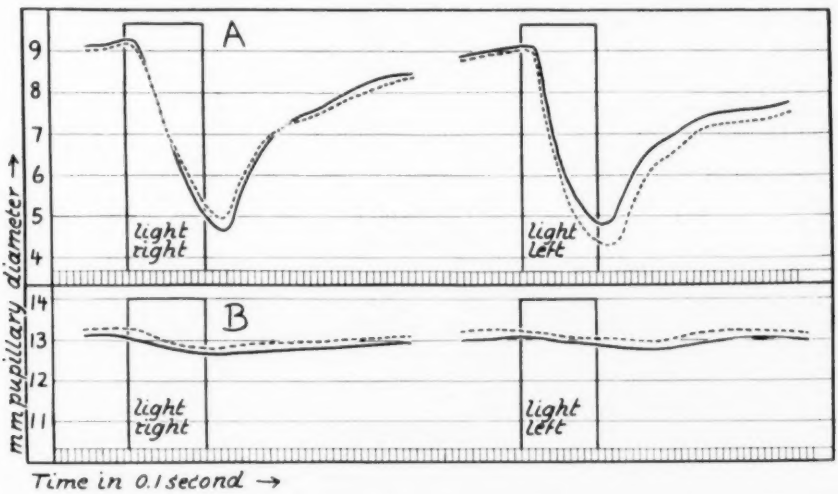

Fig. 8.—Reactions to light in the same cat as that used in Figure 7. *A*, before and, *B*, after a midline lesion in the posterior commissure.
The solid line represents the right pupil; the broken line, the left pupil.

third-nerve lesions, extent and speed of dilation may be reduced, owing to the powerful contraction of the antagonistic sphincter muscle; in destructive lesions, the already mydriatic pupils dilate in response to psychosensory stimuli within the limits of mechanical possibility.

(*b*) Lesions of the efferent third-nerve fibers central to the ciliary ganglion are functionally identical with nuclear lesions. Identical pupillograms are obtained, since in both conditions destruction affects the same neuron.

(*c*) Lesions in the ciliary ganglion result in functional effects which differ in cats from those in primates. This is due to the fact that in cats postganglionic sympathetic fibers from the superior cervical ganglion do not pass through the ciliary ganglion, as they do in man and monkeys. In cats, therefore, removal of the ciliary ganglion causes defects of the parasympathetic supply of the iris only,

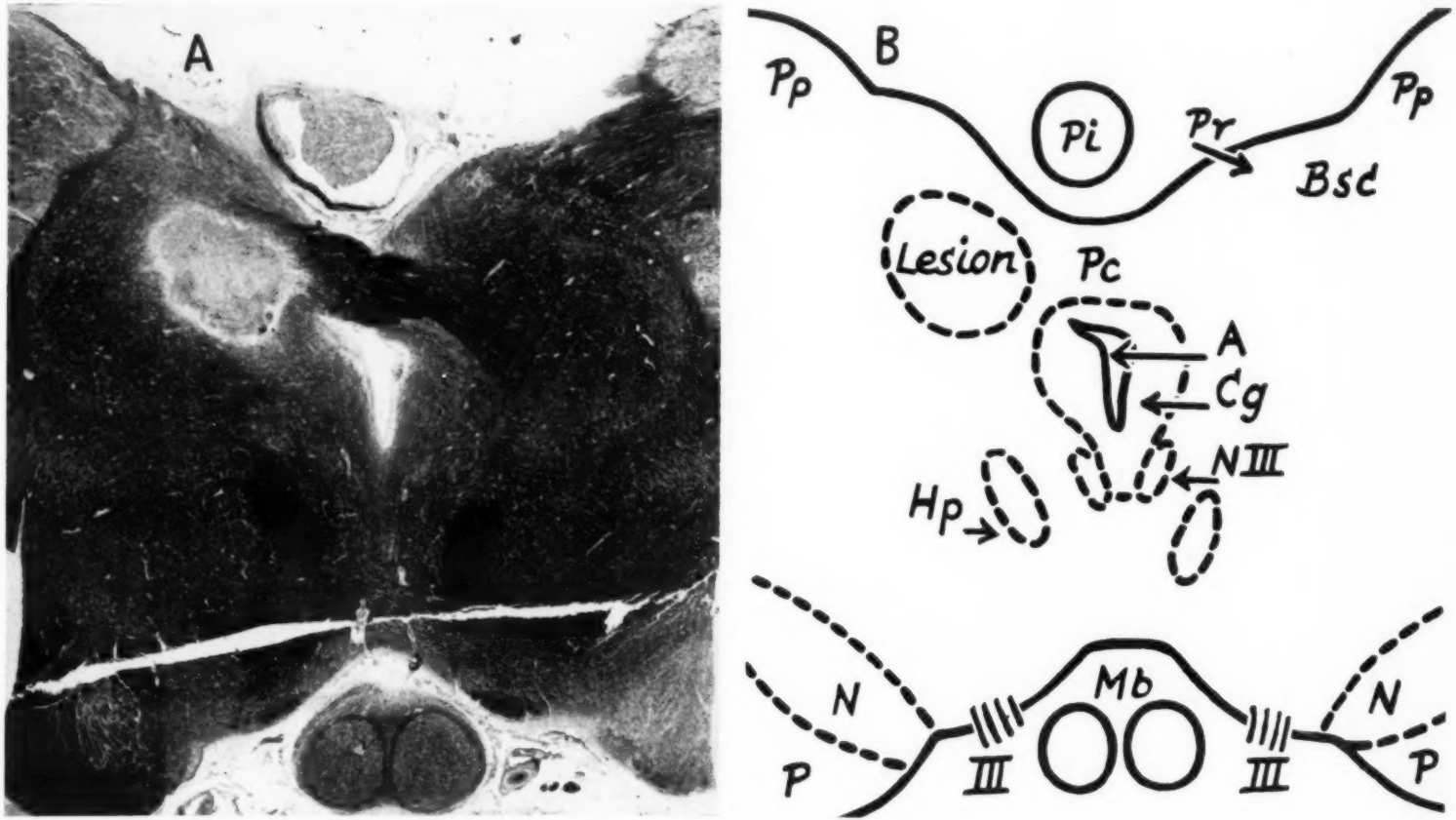

Fig. 9.—Cross section of the midbrain of a cat at the level of the posterior commissure.
A: Microscopic section (Davenport silver stain). The unilateral lesion includes all fibers coming from the left pretectal area.
B: Outline drawing of the section shown in *A*.
A indicates aqueduct of Sylvius, extending ventrally into the third ventricle; *Bsc*, brachium of superior colliculus; *Cg*, central gray matter; *Hp*, habenulopeduncular tract; *Mb*, mamillary bodies; *N*, substantia nigra; *NIII*, third-nerve nucleus; *III*, third-nerve roots; *P*, cerebral peduncles; *Pc*, posterior commissure; *Pi*, pineal body; *Pp*, posterior part of putamen; *Pr*, pretectal region.

while in man and monkeys the sympathetic supply is also involved. This results in characteristically different pupillograms. In cats, ciliary lesions have the same functional effect as lesions of the efferent third-nerve fibers; the pupils of the normal and the damaged side are equal in darkness. In man and monkeys, however, the pupil on the side of the damaged ciliary ganglion is smaller than the pupil on the normal side when the eyes are adapted to darkness. Since the light reflex is disturbed on the side of the lesion, the normal pupil becomes the smaller one upon light stimulation.

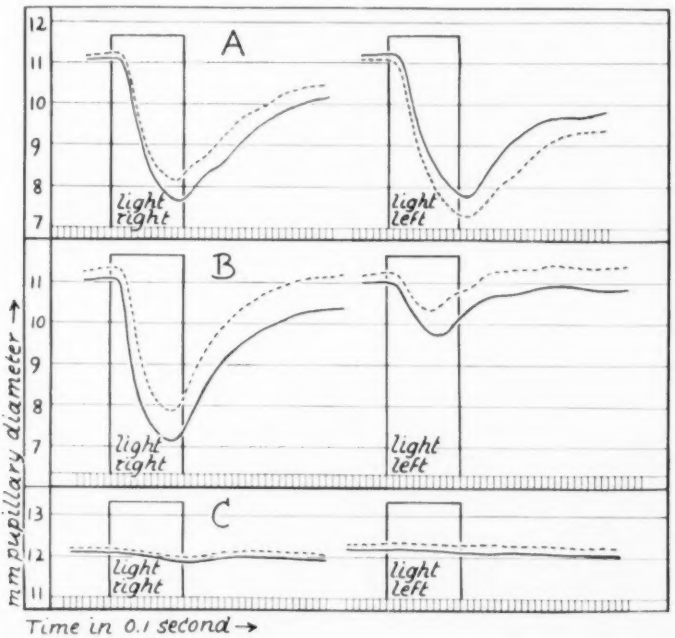

Fig. 10.—Reactions to light in a cat. The right pupil is represented by a solid line; the left pupil, by a broken line.

A: Normal responses.

B: Responses after section of right optic tract. When the right eye (crossed fibers) is stimulated, the reflex is unimpaired. When the left eye (uncrossed fibers) is stimulated, contraction is inextensive and shows a low-intensity form. When either eye is stimulated, the contraction of the right pupil exceeds that of the left pupil.

C: Responses after an additional lesion on the left side in the region of the pretectal area (Fig. 9). Direct and indirect reflexes from either eye are abolished.

SUMMARY AND CONCLUSIONS

In cats, focal lesions were produced in the various neurons which constitute the pathway of the pupillary reflex to light, and the functional effect of these lesions was quantitatively determined by pupillography.

In cats, but not in primates, the pupillomotor impulses which cross in the optic chiasm, as well as those which cross in the central decussation, from the pretectal

area to the Westphal-Edinger nucleus of the opposite side, exceed the uncrossed impulses in physiological effect. This is in agreement with the anatomical distribution of fibers in the two decussations.

The distribution of impulses from each pretectal area to the right and left Westphal-Edinger nuclei is a function of the second neuron alone. It does not depend on the quantity of impulses which reach a pretectal area or on whether these impulses arise in the retina ipsilateral or contralateral to the pretectal area which is stimulated. Therefore, contraction anisocoria is never due to lesions anterior to the pretectal area. If the efferent path is intact, it is always the result of unequal distribution of impulses in the second neuron.

Unilateral lesions in the afferent pathways always affect both pupils; unilateral lesions in the efferent pathways result in pupillary disturbances of the injured side only.

Lesions in the ciliary ganglion of cats are functionally equivalent to lesions of the efferent third-nerve fibers. In man and monkeys, such lesions include the sympathetic long ciliary fibers and therefore affect both the sympathetic and the parasympathetic factors of pupillary innervation. Therefore anisocoria due to lesions in the ciliary ganglion disappear after adaptation to darkness in cats, but not in man and monkeys.

Registry of Interesting Cases

It is the purpose of this section to record cases on the basis of interest only. No complete review of the literature is attempted and no claim for priority is implied except when specifically stated. Publication of case reports is subject to the concurrence of the Editorial Board on the interesting aspects of the case. It is requested that the reports submitted be as brief as is consistent with inclusion of the pertinent facts, bear a title that is as specific as possible, be prefaced by a paragraph that states why the author feels the case is of interest and be accompanied with photographs whenever possible.

FIBRINOID DEGENERATION OF THE TARSAL CONJUNCTIVA

THIS CASE is reported because of the unusual macroscopic and microscopic appearance of the mass, simulating a tumor, in the tarsal conjunctiva.

A woman aged 20 was first seen at the Ophthalmic Hospital of Dr. A. Ticho in November, 1951, with the complaint of impaired vision in both eyes. She had been treated for conjunctivitis in early childhood. Since that time she had rarely used eye drops or ointment.

Examination revealed an old trachoma, Stage IV in MacCallan's classification. The conjunctiva of both upper lids showed superficial scarring. The upper tarsi were slightly thickened. The conjunctiva of the lower lids was transparent, and there was a slight shortening of the lower fornix in both eyes. The cornea presented superficial maculae. Visual acuity with correction was 6/15 in each eye. The media were clear and the fundi normal.

The patient was seen again in May, 1952. At this time she complained of a foreign body sensation in both eyes, of a few weeks' duration. She had noticed something unusual on the inner surface of each lower lid. On examination a yellowish mass was seen protruding from the conjunctiva of the lower tarsus of the right eye (Fig. 1*A*). The mass, the borders of which were somewhat irregular, extended from the outer angle to the middle of the lid; its surface was uneven—in places smooth, in others nodular. It measured 16 by 4 mm.; the distance from the lid margin was about 1 mm., and that from the fornix, 4 mm. The conjunctiva of the left lower tarsus showed a similar change (Fig. 1*B*). The lesion was smaller and extended from the outer angle to the tarsal conjunctiva of the upper lid. It measured 8 by 1 to 2 mm. in the lower lid and 4 by 1 to 2 mm. in the upper lid and was situated nearer the fornix.

At operation, performed by Dr. Ticho, the masses were easily separated from the underlying tissue; there was no bleeding during this procedure. The postoperative course was uneventful. Healing was complete after 10 days. On the last examination, three months after operation, there was no recurrence of the lesions.

The tissue was fixed in 4% formalin and stained with hematoxylin and eosin, the Van Gieson method, Mallory's phosphotungstic acid hematoxylin, the azan-Petersen stain for collagen, the Weigert differential stain for fibrin, the Laidlow silver stain for reticulum, the periodic acid-Schiff stain, Millon's reagent for tyrosine, orange G-aniline blue, at a pH of 2.7, for histone, and Congo red and crystal violet for amyloid.

Many sections representing various levels of three tiny pieces of tissue were examined. The histological pictures of all three pieces were similar. Each piece was covered partially by a very thin layer of squamous epithelium (Fig. 2). Beneath the epithelium there was a dense homogeneous mass of collagen, in which were small slits and spaces, giving the impression of a dense network of thick collagen bundles. Here and there in a slit or space a capillary was visible. Cell nuclei were few, and in some areas they were absent. In the center of the largest piece (Fig. 3) the homogeneous collagen was broken up into irregular masses, which were gathered in clusters. Between the clusters was a rich, diffuse infiltration of lymphocytes and histiocytes. There were occasional giant cells, with 10 nuclei or more. A similar, but less cellu-

lar, infiltrate was noted at the periphery of the homogeneous collagen mass. This collagen material stained with eosin in various hues of red, ranging from dark-red to purple; with the Van Gieson stain most of it was yellow, and only a few reddish fibers were seen to be interspersed. With Mallory's phosphotungstic acid hematoxylin stain some of the homogeneous

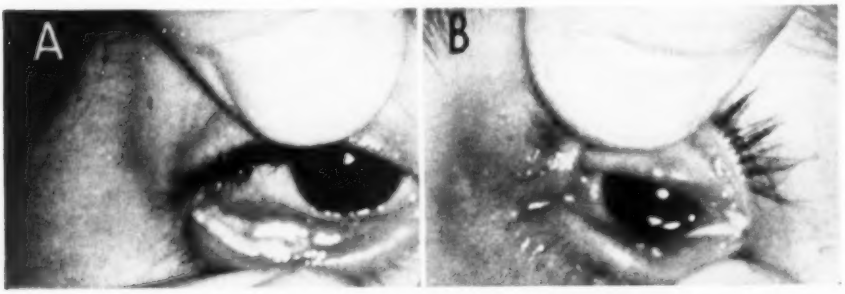

Fig. 1.—Macroscopic appearance of the lesions in (*A*) the right eye; (*B*) the left eye.

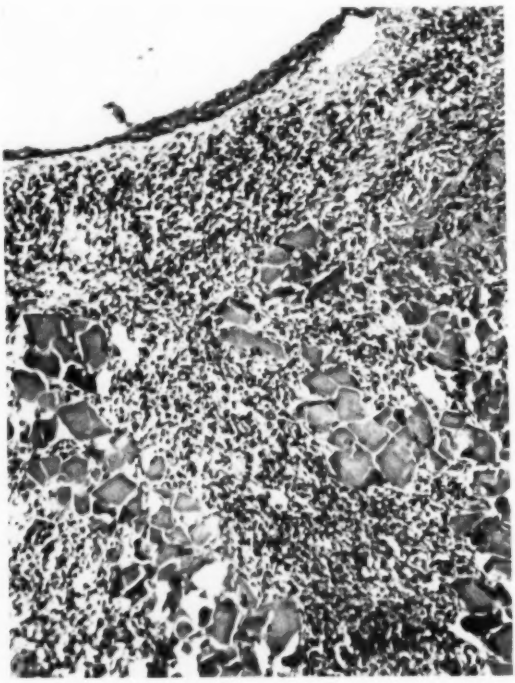

Fig. 2.—Histological appearance of the lesion covered by a thin layer of squamous epithelium. Hematoxylin-eosin stain; × 105.

collagen areas stained dark-blue, and with Weigert's differential stain for fibrin, they stained blue. Some of the areas which stained blue with Mallory's phosphotungstic acid hematoxylin, as well as with Weigert's differential stain for fibrin, stained yellow selectively with orange G-aniline blue at a pH of 2.7. All other stains, including those for amyloid, gave negative results.

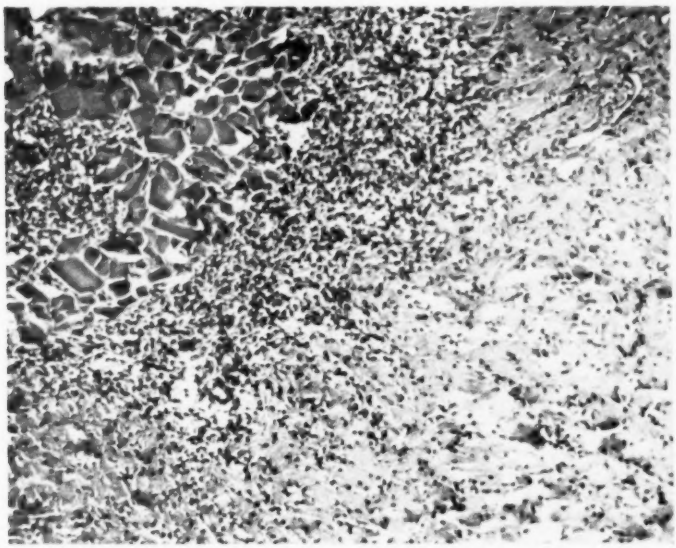

Fig. 3.—Homogeneous collagen masses at the right side; clusters of broken-up collagen on the upper left corner; diffuse rich cellular infiltration. Hematoxylin-eosin stain; × 105.

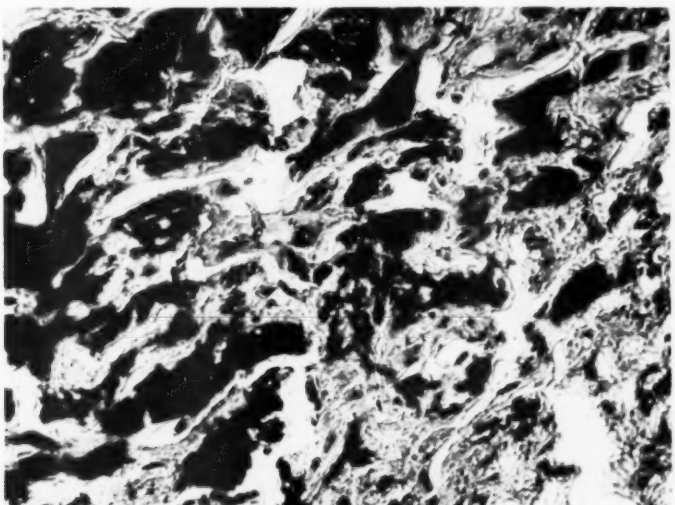

Fig. 4.—Fibrinoid material situated between altered collagen bundles. Phosphotungstic acid hematoxylin stain; × 370.

With the hematoxylin-eosin stain, a large part of the section varied in color from deep-red to purple, giving the microscopic impression of a so-called fibrinoid degeneration of collagen. This diagnosis was confirmed with the Van Gieson stain, Weigert's differential stain for fibrin, and the phosphotungstic acid hematoxylin stain. In some areas the fibrinoid material seemed to lie between the already altered collagen bundles, pushing its fibers apart. In other areas it appeared to obscure the bundles and to fade out into the surrounding collagen (Fig. 4). The selective staining with orange G and aniline blue might indicate that the fibrinoid material contained histone.[1]

One might conclude from the histological appearance that the fibrinoid material was deposited between the collagen fibers within the ground substance of the connective tissue. This finding is in agreement with the view of Altshuler and Angevine,[2] who stated the belief that the ground substance is the only constant anatomic element in the formation of fibrinoid.

This case is another instance of local fibrinoid degeneration of collagen in which the mechanism of hypersensitivity does not seem to be a factor in the pathogenesis.

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Note.—The foregoing case report and photomicrographs were submitted to Dr. F. H. Verhoeff for comment. Dr. Verhoeff pointed out that similar areas of hyaline degeneration were not uncommon in late stages of trachoma and that they have been described as variants of plasmoma.[3] To this the present authors made the following reply:

"We are in complete agreement . . . that similar histological pictures have been described as hyaline changes of the conjunctiva (or so-called plasmoma if there was a prominent plasma-cell infiltration in addition to the degenerative changes of the collagen). The microscopic appearance itself does not define adequately the nature of the alterations in the connective tissue. In order to differentiate the various forms of degeneration of the collagen, we had to do differential staining. Fibrinoid formation refers to the appearance in the connective tissue of a homogeneous, eosinophilic, refractile, relatively acellular structure, which has some of the tinctorial properties of fibrin, though it is not to be identified with it. The assumption of such a degeneration was confirmed in our case, as may be noted in the various stains used. Fibrinoid degeneration occurs in numerous lesions of various origin and pathogenesis and is, therefore, not bound to a typical histological structure. In the "comment" on our case, it is stated the fibrinoid degeneration was found not only in the aggregations but in a large part of the altered collagen bundles.

"You are probably aware that the glomerular changes in systemic lupus erythematosus, which were formerly described as hyaline degenerations, are now known to be identical with fibrinoid degeneration."

1. White, J. C.: Investigations on Some Cellular Stain Reactions, in Proceedings of Biochemical Society, Biochem. J. **46**:xxiv, 1950.

2. Altshuler, C. H., and Angevine, D. M.: Histochemical Studies on the Pathogenesis of Fibrinoid, Am. J. Path. **25**:1061, 1949.

3. Verhoeff, F. H., and Derby, G. E.: Plasmoma of the Lacrimal Sac, Arch. Ophth. **44**:252, 1915.

Annual Reviews

DISEASES OF THE RETINA

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(Concluded from Page 586)

DIABETIC RETINOPATHY

OPHTHALMOLOGISTS have been keenly interested in diabetic retinopathy since this condition was first recognized by Jaeger, in 1855.[80] As early as 1877 MacKenzie and Nettleship [81] described the characteristic clinical and pathological lesions in the retina. A period followed these observations during which clinicians thought there was no sharp clinical boundary between this disease picture in the fundus of diabetics and the changes attributed to atherosclerosis, hypertension, and renal disease. In 1935, Waite and Beetham [82] reported on the ocular abnormalities found in 2,002 diabetic patients. They stressed the occurrence of retinal vascular abnormalities in this disease and pointed out that they were proportional to the duration, rather than to the severity, of diabetes.

In recent years ophthalmologists have made fundamental contributions to the pathological nature of vascular degenerative lesions in diabetes. In studies of flat preparations of whole retinas [83] and visualization of the entire vascular tree by special stains [84] and injection techniques,[85] they have pointed out that the earliest and basic lesions in the retina are minute capillary aneurysms. Furthermore, they have shown that the majority of patients who have diabetic retinopathy also have Kimmelstiel-Wilson lesions in the kidney at autopsy.[86] Likewise, there is a marked similarity of the staining characteristics of the capillary aneurysmal dilatations of the retina and the hyalinized globular or laminated nodules in the kidney in intercapillary glomerulosclerosis.[87]

80. Jaeger, E.: Contributions to Pathology of the Eye, Vienna, k.-k. Hof und Staatsdrückerei, 1855, p. 33.

81. MacKenzie, S., and Nettleship, E.: A Case of Glycosuric Retinitis with Comments, Ophth. Hosp. Rep., London **9**:134, 1877.

82. Waite, J. H., and Beetham, W. P.: Visual Mechanism in Diabetes Mellitus: Comparative Study of 2,002 Diabetics and 457 Non-Diabetics for Control, New England J. Med. **212**:367, 1935.

83. Ballantyne, A. J., and Lowenstein, A.: I. Pathology of Diabetic Retinopathy, Tr. Ophth. Soc. U. Kingdom **63**:95, 1943; II. Retinal Micro-Aneurysms and Punctate Haemorrhages, Brit. J. Ophth. **28**:593, 1944.

84. Friedenwald, J. S.: A New Approach to Some Problems of Retinal Vascular Disease, Tr. Am. Acad. Ophth. **53**:73, 1948.

85. Ashton, N.: Injection of Retinal Vascular System in Enucleated Eye in Diabetic Retinopathy, Brit. J. Ophth. **34**:38, 1950.

86. Friedenwald, J. S.: Diabetic Retinopathy: Fourth Francis I. Proctor Lecture, Am. J. Ophth. **33**:1187, 1950. Kimmelstiel, P., and Wilson, C.: Intercapillary Lesions in the Glomeruli of the Kidney, Am J. Path. **12**:83, 1936.

87. Ashton, N.: Vascular Changes in Diabetes with Particular Reference to Retinal Vessels: Preliminary Report, Brit. J. Ophth. **33**:407, 1949.

Capillary aneurysms per se are not unique in the diabetic process.[88] They are found in venous occlusion, malignant hypertension, pernicious anemia, chorioretinitis, and in the extreme periphery of some normal retinas; but these lesions differ as to number, size, morphological appearance, and location from those of diabetic retinopathy.

During the past year, Becker [88] and Friedenwald [89] made still further contributions to the understanding of vascular degenerative lesions in diabetes. They noted that diabetic retinopathy had been reported to have appeared in diabetic patients during pregnancy, in the absence of hypertension, and to have disappeared after delivery. This suggested to them that increased endogenous adrenocorticotropic hormone during pregnancy might play a role in the pathogenesis of diabetic retinopathy. This hypothesis was further substantiated by the appearance of lesions resembling capillary aneurysms in the fundi of two patients who were receiving intravenous injections of corticotropin for sarcoid, and the disappearance of these lesions following cessation of corticotropin therapy.

Rich [90] recently found lesions resembling those described by Kimmelstiel and Wilson in the kidney of a patient receiving corticotropin intravenously. Furthermore, Becker [88] observed clusters of capillary aneurysms at autopsy in flat preparations of the retina in some cases of adrenal hyperplasia.

Previous attempts to produce diabetic retinopathy in experimental animals with alloxan alone have been unsuccessful.[91] However, Lukens and Dohan [92] reported intercapillary glomerulosclerosis in the kidneys of a dog made diabetic for five years by injection of anterior pituitary extract. Likewise, Rich and associates [93] noted the development of Kimmelstiel-Wilson-like lesions of the kidney in rabbits which had been given cortisone. Woods [94] noted similar lesions in rabbits after the administration of hydrocortisone (Compound F). No retinal lesions were apparent in these animals. On the basis of these observations, Becker [88] made rabbits diabetic with alloxan and gave them injections of corticotropin. An ophthalmoscopic picture resembling early diabetic retinopathy was found to develop in these animals. Examination of flat preparations of the retina of some of these rabbits revealed definite capillary aneurysms, as well as distortion of the capillary pattern. These findings closely simulate early diabetic retinopathy, but at the time of writing none of the animals had advanced manifestations.

Becker [88] studied the adrenal glands obtained at autopsy from a group of diabetic patients with and without Kimmelstiel-Wilson disease to determine whether there was any indication of the amount of cortisone secreted by these patients during life.

88. Becker, B.: Diabetic Retinopathy, Ann. Int. Med. **37**:273, 1952.

89. Friedenwald, J. S.: Diabetic Retinopathy, J. A. M. A. **150**:969, 1952.

90. Rich, A. R., cited by Becker.[88]

91. Janes, R. G.; Bounds, G. W., Jr., and Leinfelder, P. J.: Ocular Complications in the Rat Made Diabetic with Alloxan, A. M. A. Arch. Ophth. **48**:414, 1952.

92. Lukens, F. D. W., and Dohan, F. C.: Experimental Pituitary Diabetes of 5 Years' Duration with Glomerulosclerosis, Arch. Path. **41**:19, 1946.

93. Rich, A. R.; Berthrong, M., and Bennett, I. L., Jr.: Effect of Cortisone upon Experimental Cardiovascular and Renal Lesions Produced by Anaphylactic Hypersensitivity, Bull. Johns Hopkins Hosp. **87**:549, 1950.

94. Woods, A. C., cited by Becker.[88]

He found that the average weight of the adrenal glands for a group of 22 patients with proved Kimmelstiel-Wilson lesions of the kidney was 24% higher than that for a group of 20 patients without Kimmelstiel-Wilson lesions. Histological examination revealed an apparent correlation of the lipid-laden, vacuolated cells in the zona fasciculata of the adrenal cortex with the Kimmelstiel-Wilson lesions in the kidney. In diabetic patients without Kimmelstiel-Wilson lesions there was a markedly lower incidence of lipid vacuoles in the adrenal cortex than in the cortex of the nondiabetic patients or in the cortex of diabetic patients with Kimmelstiel-Wilson lesions. It is known that the weight of the whole adrenal glands at autopsy and the vacuolated appearance of the adrenal cortex seen at autopsy can be influenced by many factors. One, and the most important, is the cause of death. Severe infections can produce a nonvacuolated adrenal cortex, whereas sudden death is more likely to result in a vacuolated adrenal cortex. When cases of diabetes in this study were reviewed as to cause of death, the changes in the adrenal glands noted in the two groups could not be explained by this factor. The author points out that speculation at this early stage of his studies is admittedly hazardous, but he hypothesizes that in the patient with uncomplicated diabetes there may be a compensatory decrease in the capacity of adrenal cortex for certain activities, whereas in the patient in whom Kimmelstiel-Wilson lesions or retinal vascular lesions develops, the normal or relatively excessive capacity of the adrenal cortex may be related to the causation of these lesions.

Dana and his co-workers,[95] in an interesting study on 190 diabetic patients, noted a marked clinical difference between patients with and patients without Kimmelstiel-Wilson lesions of the kidney. In 57 patients with glomerular nodules diabetic acidosis was rare, in contrast to its common occurrence in 133 controls. It was likewise noted that 90% of the patients with renal lesions showed retinal microaneurysms, whereas they were present in only 10% of the controls.

It was reasoned that in patients with retinal aneurysms and hyperglycemia diabetic acidosis would not develop when their daily insulin was withheld. A study was made of five patients with retinal aneurysms whose fasting blood sugar concentrations were in excess of 200 mg. per 100 cc. and whose daily insulin dose was 15 to 30 units. When use of insulin was discontinued for three weeks, none of them gave a positive test for acetone or diacetic acid in their urine. Since the insulin dose in these patients was not large, a patient with a 12-year history of glycosuria whose hyperglycemia was uncontrolled in spite of the daily use of 50 to 130 units of insulin and who had capillary aneurysms in the retina was withheld from insulin for 30 days. The urine was examined four times daily for acetone and diacetic acid, but no ketone bodies were found during the period of study. It was concluded that there was probably a specific association of capillary aneurysms in the retina, nodules of the glomeruli, and hyperglycemia without acidosis.

Harvey, in a discussion of this work, pointed out that corticotropin and cortisone, after prolonged administration, will produce hyperglycemia, but not acidosis, in patients.

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95. Dana, G. W., and Eversole, S. L., and Zubrod, C. G.: Hyperglycemia and Its Relationship to Diabetic Retinitis and Glomerular Nodules: A Preliminary Report, Bull. Johns Hopkins Hosp. **90**:98, 1952; Clinical Entity of Retinal Aneurysms, Glomerular Nodules and Hyperglycemia, ibid. **90**:323, 1952.

Kinsell [96] and his co-workers noted a marked diminution in fasting-induced ketonuria and hyperketonemia in patients with and without Addison's disease as a result of the administration of cortisone.

Becker [88] concludes that these findings are additional evidence that the difference between diabetics with and diabetics without Kimmelstiel-Wilson lesions is a relative excess of certain adrenocortical secretions in the former group or a deficiency in the latter.

Heinsius [97] also believes that retinal vascular lesions in diabetes are not caused solely by hyperglycemia but are probably related to some other endocrine dysfunction. He does not think, however, that the glomerular lesions in the kidneys of diabetic patients are comparable to those that appear in the retina.

The aforementioned findings of Dana and his co-workers on the relationship of capillary aneurysms in the retina, nodules in the glomeruli, and hyperglycemia and acidosis may prove to be of statistical significance, but the finding is not constant. I have observed at least four patients with juvenile diabetes of 15 to 20 years' duration at the Stanford University Hospital who have had advanced diabetic retinopathy and who have frequently been in diabetic coma with acidosis. Kane [98] reports a similar case from the New York Hospital. Root and his co-workers [99] in numerous articles have stressed that patients with poorly controlled diabetes who have been in diabetic coma one or more times have a higher incidence of vascular lesions than patients whose diabetes is excellently controlled and who have never been in coma.

These studies on the basic factors involved in the development of vascular degenerative lesions in diabetes are extremely interesting, but they are still in a theoretical stage. Of more practical importance at this time is the question of the best method of handling the hyperglycemia of the diabetic patient. Almost everyone agrees that the diabetes should be kept well controlled at all times, that the patient should be kept asymptomatic and without notable glycosuria or hyperglycemia; but there are some who believe that a trace of sugar in the urine and slight hyperglycemia are not harmful. Again, this year, arguments have been presented for and against the theory that a diabetic patient should be maintained as far as possible in a state of normal glycemia and sugar-free urine at all times.

Ricketts [100] points out that the treatment of diabetes mellitus has four principal objectives: (*a*) the relief of symptoms, (*b*) the maintenance of normal nutrition, (*c*) the preservation of the insulin-producing capacity of the pancreas, and (*d*) the prevention or minimizing of complications. In most cases only a rudimentary knowledge of diabetes and insulin will enable the physician to relieve symptoms. The maintenance of normal nutrition is not much more difficult, granted the coopera-

96. Kinsell, L. W.; Margen, S.; Michaels, G. D., and Partridge, J.: Proceedings of the Second Clinical ACTH Conference, Philadelphia, The Blakiston Company, Vol. 1, 1951, p. 308.

97. Heinsius, E.: Retinitis Diabetica: Role of Endocrine Function and of Vegetative Nervous System in Its Etiology, Am. J. Ophth. **35**:861, 1952.

98. Kane, E. G.: Diabetes Mellitus: Practitioner's Conference, New York Med. (No. 15) **8**:16, 1952.

99. (*a*) Wilson, J. L.; Root, H. F., and Marble, A.: Controlled vs. Free Diet Management of Diabetes, Diabetes **1**:33, 1952. (*b*) Keiding, N. R.; Root, H. F., and Marble, A.: Importance of Control of Diabetes in Prevention of Vascular Complications, J. A. M. A. **150**:964, 1952.

100. Ricketts, H. T.: Modern Treatment of Diabetes Mellitus, J. A. M. A. **150**:959, 1952.

tion of the patient. The author states that there is evidence, both clinical and experimental, that a state of constant hyperglycemia may be detrimental to the beta cells of the islets of Langerhans. For this reason, in diabetic patients hyperglycemia should be avoided as far as possible.

The complications to which the diabetic is prone and which are either the direct result of or are favored by excess sugar in the blood and urine are infections of the urinary tract, cataract, coma, neuritis, and vascular degenerative lesions. Urinary tract infections are more frequent when the urine has a high sugar content. Cataract, especially in young people, is thought to be related to the degree of hyperglycemia. Coma, although not caused by a high blood sugar level alone, rarely occurs without it. The cause of neuritis and premature vascular disease in the diabetic is not known, but the author thinks that these complications occur more frequently in the patient with severe, poorly controlled diabetes than in the patient with well-controlled diabetes.

Keiding, Root, and Marble [99b] have extended their observations on the importance of control of diabetes in the prevention of vascular complications. Previously they had reported the results of a study on 247 patients with the onset of diabetes under the age of 30 and with a duration of illness of 10 to 34 years. In the present communication, they have included an additional 204 patients with diabetes of this type. Their criteria for excellent, good, fair, and poor control are as described in last year's review.[101] Their results indicated again that the incidence of retinopathy, arterial calcification, and nephropathy is related not only to the duration of the diabetes, but also to the degree of its control. Thus, in a series of patients with diabetes of 20 years' duration or more, moderate or marked retinopathy, arterial calcification, and nephropathy were found in 54% of those with excellent and good control, 73% of those with fair control, and 92% of those with poor control.

Tolstoi and Dolger,[102] on the other hand, believe in a more liberal management of the diabetic patient. They state that the term "free" diet is misleading and that a better term would be "normal" diet. It is their opinion that diabetic patients should maintain an optimal weight level at all times, and that they should be free of symptoms and ketosis. Insulin should be administered when a patient has hyperglycemia, and excessively high blood sugar levels should be prevented. It is not necessary, however, to maintain normal glycemia or to prevent the spilling of some sugar in the urine. They state that patients controlled in this manner are not comparable to experimental animals, in which excessive hyperglycemia, produced by the injection of dextrose, caused damage to the beta cells of the pancreas. Likewise, it is their opinion that mild hyperglycemia and glucosuria do not cause vascular degenerative lesions in the diabetic patient.

The theory of these authors on the management of diabetes is supported in some degree by a study by Bell [103] of the vascular lesions seen at 1559 autopsies on diabetic subjects. In about 5% of this group of diabetic patients, the disease

101. Maumenee, A. E.: Diseases of the Retina (Annual Review), A. M. A. Arch. Ophth. **47**:643, 1952.

102. Tolstoi, E., and Dolger, H.: Diabetes Mellitus: Practitioner's Conference, New York Med. (No. 15) **8**:16, 1952.

103. Bell, E. T.: A Postmortem Study of Vascular Disease in Diabetics, A. M. A. Arch. Path. **53**:444, 1952.

developed before the age of 20, in 16% before the age of 40, and in 70% after the age of 50. There were no deaths from vascular disease before the age of 20; between the ages of 20 and 40 about 23% of the deaths were due to vascular disease, and after the age of 50 about 54% were from vascular lesions. In a control study of nondiabetic patients, about 30% of subjects over 50 years of age died of some form of vascular disease; therefore only about 24%, and not 54%, of the deaths among the older diabetics should be attributed to the diabetic state. The types of vascular disease especially accelerated by diabetes were gangrene, renal arteriosclerosis, and coronary disease.

When the onset of diabetes was before the age of 40, the influence of the duration of the disease on the development of vascular lesions was striking. Few persons presented vascular lesions during the first 10 years of diabetes, but after 20 years or more only a few escaped. However, vascular disease did not invariably develop after 20, or even 30, years. In 28 of 93 persons who lived more than 20 years after the onset of diabetes, necropsy revealed no serious vascular disease. In these 28 patients, the absence of vascular disease could not be attributed to better control of the diabetes. In the older diabetics there was no relation between the severity of the disease and the tendency toward vascular disease.

In seven patients having diabetes associated with hemachromatosis, of ages ranging from 51 to 66, no vascular complications were seen at autopsy. The duration of the diabetes ranged from four months to six years. The absence of vascular lesions might have been due to the short duration of the diabetes, or "bronze diabetes" may have a different cause. Lawrence [104] found no vascular complications in 40 cases of diabetes with hemachromatosis.

Johnson and Rynearson [105] report a diabetic patient who was maintained on a high-fat diet and insulin for 29 years without vascular complication. Bottoni [106] observed the action of insulin on the ocular fundus of 236 diabetic patients, 147 of whom with known diabetes for 1 to 30 years had some evidence of diabetic retinopathy; 3 had retinitis proliferans. The author concludes that insulin was not the cause of diabetic retinopathy and that the best therapy known at this time is adequate control of the diabetes.

Niedermeier [107] found that cysteine (Hormodyn) therapy in diabetic patients had no influence on the development of punctate hemorrhages.

Root [108] reported 326 cases of retinitis proliferans, in 114 of which the onset of diabetes was prior to the age of 20 and in 86 of which diabetes developed between the ages of 20 and 39. Thus, in almost two-thirds of cases severe diabetes developed in youth, although less than one-third of all cases of diabetes develop in this age group. Females were affected more frequently than males, but only slightly so. Retinitis proliferans is a poor prognostic sign, for more than one-third of the patients died of renal failure or uremia within three years after its development.

104. Lawrence, R. D.: Hemochromatosis and Complications of Diabetes, Physicians Bull. **12**:34, 1947; Vascular Changes in Diabetes, Brit. M. J. **2**:107, 1950.

105. Johnson, H. W., and Rynearson, E. H.: A Diabetic Patient on High Fat Diet for 29 Years Without Complications, Proc. Staff Meet., Mayo Clin. **26**:329, 1951.

106. Bottoni, A.: Diabetic Retinopathy and Insulin Therapy, Gior. ital. oftal. **5**:408, 1952.

107. Niedermeier, S.: Therapy of Diabetic Retinitis, Klin. Monatsbl. Augenh. **121**:559, 1952.

108. Root, H. F.: Diabetic Eye Conditions, Sight-Saving Rev. **22**:103, 1952.

Handelsman and his co-workers [109] studied the skin-temperature response in the toes of diabetic patients to an intravenous injection of 2 cc. (50 mg.) of benzazoline (Priscoline). In 7 of the 15 patients studied, vasodilatation, as measured by increase in temperature of the toes, was poor. The authors conclude that, in addition to the occurrence of well-recognized "small-vessel disease" in the retina and kidneys of diabetics, this type of vascular damage also occurs in the extremities of diabetics. This small-vessel dysfunction exists in the presence of clinically normal peripheral circulation.

In the same clinic, Cholst and his co-workers [110] studied the influence of the administration of benzazoline on the widening of the angioscotoma in diabetic patients. In 12 of the 28 patients the widening of the scotoma was graded as poor. This response was more closely correlated with hard exudates than with punctate aneurysms in the retina. Six of the patients in this series had had parallel studies on the temperature response in their toes following the injection of benzazoline, but no correlation was found between the "small-vessel" dysfunction in the retinal vessels and that shown to occur in the skin of the toes.

Givner [111] makes a plea for a standardization of the recording of data on diabetic patients so that a larger amount of comparable information may be collected from various clinics. He states that in a series of 28 patients who had had known diabetes for 25 years or longer, 21, or 75%, still had 20/40 vision or better. All these patients showed some evidence of diabetic retinopathy, and in 14% there were advanced cataractous changes. Two per cent had detachments of the retina, and 3% had retinitis proliferans.

Cordes,[112] in an extensive review of the literature, discusses the visual prognosis of the diabetic patient. He states that the young diabetic who survives 20 years is likely to have a severe form of diabetic retinopathy, with loss of vision, regardless of the level of control at which the disease has been maintained. He points out, however, that recent scattered reports indicate that if present-day methods of control are carried out meticulously, the prognosis in the future may not be as black as it is now generally believed to be.

Marr [113] studied the fundi of 61 juvenile diabetics. Thirty-seven of these had had diabetes for less than 10 years, and 2 showed advanced retinopathy. Of 24 patients who had had diabetes for 10 years or longer, there were 13 with diabetic retinopathy. He concludes that the duration of the diabetes is paramount in the development of the characteristic retinal lesions.

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109. Handelsman, M. B.; Levitt, L. M., and Conrad, H. Jr.: Small Vessel Dysfunction in Patients with Diabetes Mellitus: I. Skin Temperature Response to Priscoline in the Toes of Diabetics, Am. J. M. Sc. **224**:34, 1952.

110. Cholst, M. R.; Levitt, L. M., and Handelsman, H. P.: Small Vessel Dysfunction in Patients with Diabetes Mellitus, II. Retinal Vessel Response in Diabetics Following Priscoline, Am. J. M. Sc. **224**:39, 1952.

111. Givner, I.: A Group Study of Diabetes and the Eye, Am. J. Ophth. **35**:1454, 1952.

112. Cordes, F. C.: The Diabetic: His Visual Prognosis, A. M. A. Arch. Ophth. **48**:531, 1952.

113. Marr, W. G.: Cataracts and Retinopathy in Juvenile Diabetics, Am. J. Ophth. **35**:577, 1952.

Everett [114] describes a case of nondiabetic lipaemia retinalis in a boy 3½ years of age. The child had had numerous generalized convulsions which could not be accounted for by any detectable cerebral abnormality. Four and one-half months before lipaemia retinalis developed, he had been placed on a high-fat diet with a ketogenic ratio of 4 : 0. The diet contained 84% fat by weight. The lipaemia retinalis disappeared after 24 hours of fasting, although the serum remained milky throughout the patient's stay in the hospital.

Lewis [115] describes the histological examination of an eye which she had previously reported as showing nondiabetic lipaemia retinalis. Ophthalmoscopic examination revealed widely scattered yellowish patches throughout the fundus. The largest mass formed an extensive tumor involving the lower one-sixth of the fundus, producing a detachment of the retina over it. Histological examination revealed the large mass to be connective tissue, separating the retina from the choroid. Xanthoma cells were present in the retina and choroid, as well as at the periphery of the granulation tissue. The center of the large mass was composed of cholesterol crystals. Frozen sections stained with Sudan III showed a characteristic reaction for fat in the cells in the retina, choroid, and granulation tissue. These sections showed all the changes of primary essential xanthomatosis.

TOXEMIA OF PREGNANCY

Landesman and his co-workers [116] studied the retinal changes in toxemia of pregnancy in relation to mild and severe hypertension, renal disease, and diabetes mellitus. They draw the following conclusions: 1. Retinal changes in hypertensive disease are, in general, more advanced than those found in acute toxemias because of previous arteriolar disease. 2. Early retinal changes in mild hypertension are of little prognostic significance but are of great value in providing a basis for the interpretation of future developments. 3. In severe hypertension the character of the retinal arterioles may have great prognostic significance. The percentage of pregnancies requiring interruption increases rapidly with the degree of retinal arteriolar damage in severe hypertension. The presence of retinal hemorrhages, transudates, or papilledema associated with hypertension is an indication for the prompt interruption of pregnancy regardless of the duration of gestation. 4. In chronic renal disease, the prognosis for a living infant decreases as the severity of retinal vascular changes increases. 5. In diabetes mellitus associated with toxemia, retinal vascular changes are a more ominous prognosis for the fetus than in toxemia alone. Grade 2 arteriolar changes in the retina of a diabetic patient usually indicate death of the fetus unless early intervention of pregnancy is performed. 6. Diabetic retinopathy is a hazard to the vision of the mother. This complication tends to increase in severity during pregnancy and may seriously involve the macular area. Patients with diabetic retinopathy should not attempt pregnancy.

114. Everett, W. G.: Nondiabetic Lipemia Retinalis: Report of a Case, A. M. A. Arch. Ophth. **48**:712, 1952.

115. Lewis, N.: Case Notes: Ocular Pathology in a Case of Xanthomatous Biliary Cirrhosis with Intra-Ocular Involvement, Brit. J. Ophth. **36**:325, 1952; Intra-Ocular Involvement in a Case of Xanthomatous Biliary Cirrhosis, ibid. **34**:506, 1950.

116. Landesman, R.; Douglas, R. G., and Snyder, S. S.: Retinal Changes in the Toxemias of Pregnancy: II. Mild and Severe Hypertension, Renal Disease, and Diabetes Mellitus, Am. J. Obst. & Gynec. **63**:16, 1952.

Snyder,[117] on the basis of the clinical material just cited, reports in more detail on the retinal changes in pregnancy complicated by diabetes mellitus and toxemia. His conclusions are essentially the same as those noted above.

RETINAL CHANGES IN GENERAL MEDICAL DISEASES

During the past few years ophthalmologists have shown an increasing interest in a group of conditions known as the collagen diseases, which have been defined by Klemperer [118] as clinically unrelated conditions similar only in that most of them appear to be due to a hypersensitivity reaction and all have a peculiar fibrinoid degeneration of connective tissue or ground substance. At one time, the fibrinoid degeneration was thought to be due to changes in the collagen fibers; thus the name for the condition. It is now thought, however, that the fibrinoid tissue response represents an altered or precipitated protein-mucopolysaccharide substance which is laid down by altered connective tissue cells between the collagen fibers. Some of the conditions which fall into this group are lupus erythematosus, scleroderma, dermatomyositis, serum sickness, allergy to sulfonamides, periarteritis nodosa, and rheumatoid arthritis.

The pathological and clinical manifestations of these diseases are described in some detail by Keefer [119] and Klemperer.[120] Ehrich [121] reports the histochemical changes in the tissues and discusses the possible pathogenesis of the collagen diseases.

The scleral and uveal changes which are associated with this type of pathological change will probably prove to be of greatest interest to ophthalmology in the future, although it has been known for a number of years that retinal changes do occur during the acute stages of some of the collagen diseases.

Wise [122] reviewed the literature on the ocular manifestations of periarteritis nodosa and reports two cases in which scleral, corneal, uveal, and retinal lesions were present. Goar and Smith [123] describe two patients with periarteritis nodosa in whom the eyes were examined both clinically and histologically. The retinas showed edema, transudates, hemorrhages, and cytoid body formation. The arteries showed areas of focal degeneration with fibrinoid changes in the walls and perivascular inflammatory-cell infiltration.

Aiello [124] reports the case of a 12-year-old boy with lupus erythematosus whose initial diagnosis was infectious mononucleosis. The first ocular signs of the disease were cotton-wool patches throughout both fundi, blurring of the disk margins, and edema of the retina. At a later stage, hemorrhages and further exudates developed in both fundi. The child was given both corticotropin and cortisone, which produced

117. Snyder, S. S.: Retinal Findings in Pregnancy Complicated by Diabetes Mellitus and Toxemia, Am. J. Ophth. **35**:831, 1952.

118. Klemperer, P.: Collagen Disease, Editorial, Am. J. Med. **10**:405, 1951.

119. Keefer, C. S.: Collagen Diseases: Pathology and Clinical Course, M. Clin. North America. September, 1951, p. 1267.

120. Klemperer, P.: Concept of Collagen Diseases, Am. J. Path. **26**:505, 1950.

121. Ehrich, W. E.: Nature of Collagen Diseases, Am. Heart J. **43**:121, 1952.

122. Wise, G. N.: Ocular Periarteritis Nodosa: Report of 2 Cases, A. M. A. Arch Ophth. **48**:1, 1952.

123. Goar, E. L., and Smith, L. S.: Polyarteritis Nodosa of the Eye, Am. J. Ophth. **35**: 1619, 1952.

124. Aiello, J. S.: Ocular Findings in Lupus Erythematosus, Am. J. Ophth. **35**:837, 1952.

a remission of his symptoms. The patient died five months after onset, and the diagnosis of lupus erythematosus was confirmed by necropsy. Histological study of the eyes revealed scattered small hemorrhages in the nerve fiber layer, cytoid bodies in the retina, sclerosis of the retinal vessels, with a slight amount of perivascular round cell infiltration, but no evidence of necrotizing lesions in the vessel walls.

The ophthalmic manifestations of temporal arteritis are discussed by Parsons-Smith.[125] He points out that this disease characteristically attacks patients between the ages of 55 and 85. It is usually benign, self-limiting, and insidious in development, with a course lasting over several months. In a few instances the disease has resulted in death. As the name implies, a regular feature is the inflammation of the temporal arteries of the face, but the lesion is not always confined to these vessels, and any of the cranial arteries may be involved.

The pathological features are those of a subacute inflammatory process commencing in the adventitia and later involving the media and intima. In the smaller vessels thrombosis is common as a sequela. The optic nerve or retina was involved in 9 of 15 patients reported by the author. In eight of these patients the visual acuity in one or both eyes was moderately or severely impaired. No specific treatment yet exists for temporal arteritis, but it is suggested that use of heparin or other anticoagulant therapy may be of value during the acute stage of the disease.

The origin and pathological nature of cytoid bodies in the retina is discussed by Kiewe and Hart.[126] The material used in their study were retinas from patients with bronchogenic carcinoma, choroidal melanoma, lupus erythematosus, diabetes, and hypertension. Routine stains confirmed all the well-known characteristics of cytoid bodies, but staining methods for neuroglia failed to give the results required for glial identification. The Hotchkiss-McManus staining technique for polysaccharides showed a picture very similar to that produced by hematoxylin and eosin, namely, a dark, irregular center reminiscent of a cell nucleus and a slightly less intensely staining periphery. The Feulgen staining technique and iron hematoxylin stains, however, failed to reveal evidence of nucleoproteins. This result was interpreted as indicating the non-nuclear nature of the dark centers. Scarlet-red staining of whole pieces of retina and of Carbowax sections failed to show fat or lipids. The authors conclude that cytoid bodies are not cellular in origin, but are probably exudative lesions in the tissues consequent to capillary damage. The peculiar shape of the cytoid body, which has been attributed to a ganglioform, varicose degeneration of the nerve fibers, is due to the molding of trapped exudates within the supportive network of the nerve fiber layer.

The changes in the eye and orbit following carotid ligation are discussed by Swan and Raaf.[127] Five patients with a fistula of the carotid artery into the cavernous sinus were treated by simultaneous ligation of both external and common carotid arteries on the affected side. In three of these patients a severe retinopathy developed several days after operation. This was characterized by cyanosis of the

125. Parsons-Smith, G.: Ophthalmic Manifestations of Temporal Arteritis, Brit. J. Ophth. **36**:615, 1952.

126. Kiewe, P. L., and Hart, F. D.: Cytoid Bodies in the Retina in a Case of Malignant Disease, Tr. Ophth. Soc. U. Kingdom **71**:309, 1951.

127. Swan, K. C., and Raaf, J.: Changes in the Eye and Orbit Following Carotid Ligation, Tr. Am. Ophth. Soc. **49**:435, 1951.

retina, macular edema, and multiple cotton-wool patches. The retinopathy gradually cleared in two of the patients, leaving only focal areas of capillary dilatation and aneurysmal formation; but hypotony, retinal edema, and hemorrhages have persisted for six months in the third patient.

Adler,[128] in a discussion of this report, states that the fundus lesions were suggestive of hypertensive retinopathy and that the mechanism for producing the retinopathy was probably anoxia. He suggests that the lowering of the blood pressure and narrowing of the arteries produced slowing of the blood flow, which, when sufficiently impeded, caused dilatation of the capillaries. These then leaked protein, and hemorrhages and exudates occurred.

Walsh,[128] in discussing this problem, states that the retinal lesions were probably due to an obstructive mechanism of the blood supply to the retina. He had observed a number of patients in which the internal carotid artery alone had been tied off, but retinopathy was not a prominent feature in these patients. He concluded that the reason the retina was not affected when the internal carotid artery alone was occluded was an extensive anastomotic supply between the ophthalmic artery and the branches of the external carotid artery.

Givner [129] discusses briefly an extensive list of systemic diseases which have both ocular and cutaneous manifestations. Those which affect the retina are tuberous sclerosis, neurofibromatosis, Sturge-Weber-Krabbe disease, lupus erythematosus, dermatomyositis, scleroderma, periarteritis nodosa, Vogt-Koyanagi disease (Harada's disease), brucellosis, coccidioidomycosis, tuberculosis, syphilis, lymphogranuloma venereum, and monocytic leukemia.

Benton [130] also lists the incidence and diagnostic value of lesions of the ocular fundus in hospitalized medical patients.

Berens and his co-workers [131] suggest that the following retinal lesions may be due to a hypersensitivity to bacteria or other antigens: recurrent hemorrhage of the vitreous during adolescence, hypertension in the central artery of the retina, retinal separation, central angiospastic retinopathy, acute macular exudative chorioretinitis, and lupus erythematosus.

The case of a man aged 26 with periarteritis of the retina is described by Thompson.[132] The patient was observed over a 10-month period, during which time white plaques and "pink fluffy" areas disappeared and appeared on many of the retinal arteries. The veins were not affected. The etiology of the disease was not determined.

Jefferson and Cloake [133] reviewed the literature on Eales's disease with particular reference to neurological lesions which, in the past, have been described as accom-

128. In discussion on Swan and Raaf.[127]

129. Givner, I.: Symposium: Ocular Manifestations of Dermatologic Diseases; Associated Eye and Skin Manifestations of Systemic Disease, Tr. Am. Acad. Ophth. **56**:751, 1952.

130. Benton, C. D., Jr.: Incidence and Diagnostic Value of the Ocular Fundus Lesions in Hospitalized Medical Patients, Am. J. M. Sc. **224**:554, 1952.

131. Berens, C.; Sayad, W. L., and Girard, L. J.: Symposium: Ocular Allergy: The Uveal Tract and Retina; Considerations of Certain Experimental and Clinical Concepts, Tr. Am. Acad. Ophth. **56**:220, 1952.

132. Thompson, A. M. W.: Case Notes: Peri-Arteritis Retinae, Brit. J. Ophth. **36**:268, 1952.

133. Jefferson, M., and Cloake, P. C. P.: Nervous Manifestations of Eales's Disease, Brit. J. Ophth. **36**:143, 1952.

panying this condition. They also made a thorough study of six patients with this disease. Their conclusion was that neurological manifestations which accompany Eales's disease are probably coincidental. In one case, however, reported by Marchesani and Stauder, in which autopsy was performed, thromboangiitis obliterans was found both in the retina and in the brain, making the relationship undoubted.

Woods [134] stated that Eales's disease does not respond to cortisone therapy.

Danis and his co-workers [135] report that a patient with retinal periphlebitis responded to streptomycin therapy, and another patient, with recurrent vitreous hemorrhages, failed to respond to streptomycin. Humblet [136] states that use of streptomycin must be started early in the inflammatory process to be effective in the treatment of Eales's disease. Marked improvement was noted in four patients who were treated during the early stages, but streptomycin had no effect on two patients who were treated during the secondary stage.

Kapuściński [137] describes 11 cases of periphlebitis retinae. In eight the disease was ascribed to a focal origin, and in one it was thought to be of tuberculous origin. Of the remaining two cases, Raynaud's disease was present in one, and a probable tuberculous origin accounted for the condition in the other.

Faldi and Miani [138] report a case of a late stage of Hodgkin's disease with bilateral retinal hemorrhages, particularly in the macular area. These lesions are explained as a consequence of aplastic myelosis and a deficiency of platelets. The predilection of the hemorrhages for the macular area is explained by the peculiar vascularization of this part of the retina.

In an excellent discussion of the fundus changes in leukemia, Culler [139] states that at one time ophthalmologists frequently pointed out a probable diagnosis of leukemia to the internist. Bone marrow biopsies have now supplanted ophthalmoscopic examination in the diagnosis of this type of disease, and the ophthalmologist is no longer a primary contributor. However, he is still often asked to answer two questions: 1. Does the onset of retinal hemorrhages have any prognostic significance? 2. To what factor in the patient's condition are the fundus lesions due? After a review of the literature, in an attempt to answer these questions, Culler analyzes 144 cases of leukemia in which repeated fundus examinations had been done. The highest incidence of retinal hemorrhage was found in cases of acute myeloid leukemia; the lowest incidence, in cases of chronic monocytic leukemia. However, it must be stressed that hemorrhages in the fundus accompany all types of leukemia and occur at all levels of white cell concentration in the blood. The retinal lesions are therefore of little prognostic significance. The flame-shaped hemorrhages which usually lie in relation to a visible vein may be accepted as

134. Woods, A. C.: Uses and Limitations of ACTH and Cortisone in Ophthalmology, Brit. J. Ophth. **36**:401, 1952.

135. Danis, P.; Humblet, M., and Lambert, P.: Periphlebitis Retinae and Streptomycin, Bull. Soc. belg. opht. **100**:337, 1952.

136. Humblet, M.: Importance of the Early Treatment with Streptomycin in Iridocyclitis, Bull. Soc. belg. opht. **100**:354, 1952.

137. Kapuściński, W. J., Jr.: Etiology of Periphlebitis Retinae, Arch. ophth. **12**:170, 1952.

138. Faldi, S., and Miani, P.: Hemorrhagic Retinopathy in the Course of Severe Hodgkin's Disease, Gior. ital. oftal. **5**:51, 1952.

139. Culler, A. M.: Fundus Changes in Leukemia, Tr. Am. Ophth. Soc. **49**:445, 1951.

evidence of venous obstruction by an intramural thrombus. The cotton-wool spots are probably ischemic infarcts. The venous engorgement is probably caused by localized leucocytic infiltration of the retina. The author concludes that after elimination of certain factors, such as anemia, lymphocytosis, and lowered platelet count, leukemic retinopathy is due to leucocytic stasis and infiltration, rather than to changes in other formed elements of the blood, and that the high tissue pressure of the eye probably increases the stasis.

Rodger [140] studied 238 patients with nutritional amblyopia who had acquired this lesion while they were prisoners of war in the Far East. He divides the course and progress of the disease into three stages: the acute, the subacute, and the chronic. The acute stage corresponded to the onset of the disease; the subacute stage included the period after the end of the acute stage through the remaining months of imprisonment, and the chronic stage began after restoration of normal conditions of diet and environment. The author was able to observe the patients personally only at the end of the subacute stage and during the chronic stage. The time of onset of the amblyopia following the institution of a deficient diet varied considerably. In some instances it developed in three months, and in others, in three years. The average time of onset for the patients in this study was 11.2 months after imprisonment. The initial symptom was intermittent blurring of central visual acuity, which began insidiously. Ophthalmologists who were also prisoners of war attempted to plot changes in visual fields, and, although handicapped by lack of equipment, have reported that the patients showed central and paracentral scotomas. Most observers report that no changes in the fundus were evident on ophthalmoscopic examination, but some describe slight macular granularity, and in some instances paramacular exudates.

It is interesting that an apparent tolerance to conditions causing nutritional amblyopia developed in a number of persons while they were imprisoned, for their visual acuity did not decrease appreciably after the initial onset of their symptoms. However, when they were discharged from prison camp and placed on normal diets, approximately one-third of the patients showed progressive loss of visual acuity, approximately one-third showed improvement, and one-third showed a stationary condition.

Visual field changes characteristically were equal and bilateral and consisted of a paracentral scotoma, absolute in nature, steep-edged, and never breaking through to the periphery. This finding was present in 63.4% of the patients. The average extent was 5 degrees, and the defect was approximately circular. Cecocentral scotomas were surprisingly rare, occurring in only 1.6%. In 13 of the patients the scotomas cleared partially or completely after discharge from the prison camps. These improvements, however, bore no relation to improvement in visual acuity. The author thinks this can be explained on the basis that, although the scotoma may clear, the vital foveal area may still be affected.

In every patient in this series there gradually developed clear-cut partial or complete optic nerve atrophy. The term "atrophy of the disk" as used in this discussion refers to the ophthalmoscopic appearance of the nerve head rather than to the functional or histological condition of the nerve fibers. Macular changes were not noted on ophthalmoscopic examination.

140. Rodger, F. C.: Nutritional Amblyopia: A Statistical Report of the Course and Progress in 238 Cases, A. M. A. Arch. Ophth. **47**:570, 1952.

The author states that nutritional amblyopia is closely related to toxic amblyopia and is not due to an inflammatory process, such as has been suggested by other workers.

Vom Hofe [141] studied the ocular changes in 400 patients with tuberculosis who were confined in a sanatorium and in 400 control patients, without tuberculosis. He found that the incidence of chorioretinitis, periphlebitis, retinal hemorrhages, pigment spots, and white spots in the fundus was essentially the same in the two groups. He concludes, however, that a tuberculous etiology was probable in patients with tuberculosis of the lung who had fresh choroiditis or periphlebitis.

Chamberlain [142] discusses the ocular findings in scrub typhus. He states that the most frequent involvement of the posterior segment was in the form of edema of the optic nerve and the adjacent retina. Occasionally, small superficial, flame-shaped hemorrhages and cotton-wool exudates are noted in the retina in the vicinity of the optic nerve and the macula.

Barnes and Boshoff [143] examined ophthalmoscopically 82 patients with porphyria. Forty-two of this group had fundus lesions, frequently in the form of round, cotton-wool patches of edema located in the posterior part of the fundus. As these absorbed, they left dense, black "globular" patches of pigment if the edema had persisted for three weeks or longer. Edema of shorter duration produced a diffuse granularity, if it left any sequelae. The second type of lesion consisted of flat, thin patches of edema in the periphery of the fundus. These might leave thin scars which have a yellowish or pigmented appearance. The third type of lesion consisted of hemorrhages in the retina, preretinal, intrafibrillary, or deep. If these hemorrhages, particularly the deep ones, persisted for a long period, they left dense areas of pigmentation in the retina.

TAPETORETINAL DEGENERATION

Franceschetti and his co-workers [144] discuss the modes of inheritance and clinical manifestations of the various types of tapetoretinal degeneration. They point out that there is a marked variation in the modes of inheritance from family to family, as well as an overlapping in the clinical appearance of these lesions. Thus, any classification must be considered only as a general guide, rather than a strict differentiation of the various changes. The authors' classification of tapetoretinal degeneration is as follows:

I. Cerebroretinal affections

A. Infantile amaurotic idiocy (Tay-Sachs disease)

B. Late familial amaurotic idiocy (Bielschowsky)

C. Juvenile amaurotic idiocy (Batten and Mayou; Vogt-Spielmeyer disease)

D. Adult form of amaurotic idiocy (Kufs)

141. vom Hofe, K.: Frequency of Retinal and Choroidal Changes in Pulmonary Tuberculosis, von Graefes Arch. Ophth. **152**:581, 1952.

142. Chamberlain, W. P., Jr.: Ocular Findings in Scrub Typhus, A. M. A. Arch. Ophth. **48**:313, 1952.

143. Barnes, H. D., and Boshoff, P. H.: Ocular Lesions in Patients with Porphyria, A. M. A. Arch Ophth. **48**:567, 1952.

144. Franceschetti, A.; Klein, D.; Forni, S., and Babel, J.: Clinical and Social Aspects of Heredity in Ophthalmology, Acta XVI Cong. Ophth., London **1**:156, 1951.

II. Tapetoretinal degeneration without dementia
  A. Macular type
    1. Congenital or infantile form (Best)
    2. Juvenile form (Stargardt)
    3. Adult form (Behr)
    4. Presenile and senile forms (Tay's central guttate choroiditis; senile form of Waardenburg)
    5. Atypical forms
      (*a*) Patterned familial colloid degeneration (Holthouse and Batten's "chorioretinitis"); Doyne's honeycomb "choroiditis"; malattia levantinese (Vogt, Wagner, and Klainguti)
      (*b*) Nonpatterned type: exudative-hemorrhagic type (Sorsby)
  B. Pigmentosa type
    1. Infantile tapetoretinal amaurosis (Leber)
    2. Typical pigmentary degeneration of the retina (retinitis pigmentosa)
    3. Atypical forms
      (*a*) Central pigmentary degeneration (inverse type)
      (*b*) Retinitis pigmentosa sine pigmento
      (*c*) Unilateral forms
      (*d*) Oguchi's disease
      (*e*) Fundus albipunctatus cum hemeralopia (Lauber), stationary form
      (*f*) Retinitis punctata albescens, progressive form
      (*g*) Atrophia choroideae et retinae gyrata (Fuchs)
      (*h*) Choroideremia (Mauthner)
    4. Complicated forms
      (*a*) With deaf-mutism
      (*b*) With heredoataxias
      (*c*) With external ophthalmoplegia
      (*d*) Bardet-Biedl syndrome (imbecilitas dystrophica complicata, Monrad-Krohn disease)
      (*e*) With dystrophic skin affections

François [145] reports a family in which there were cases of typical pigmentary degeneration of the retina and cases of a rather peculiar retinal degeneration which essentially affects the pigment epithelium and is not accompanied by any functional disturbance. He emphasized the pleomorphic nature of heredodegenerative affections of the macula, not only in different families, but also in one and the same family.

Feigenbaum [146] reports a family in which juvenile idiopathic retinal detachment occurred. A father totally blind from the disease had six children, of whom three were affected.

Stergar and Esche-Duval [147] describe the case of a man aged 20 who had the Laurence-Moon-Biedl syndrome. When the patient was first observed, in 1941,

145. François, J.: New Contributions to the Study of Heredodegenerative Tapetoretinal Lesions, Peripheral and Central, Acta XVI Cong. Ophth., London **1**:498, 1951.

146. Feigenbaum, A.: Familial Detachment of the Retina and the Question of Interruption of Pregnancy Because of Juvenile Idiopathic Detachment, Acta XVI Cong. Ophth., London **1**:514, 1951.

147. Stergar, S., and Esche-Duval, L.: Dystrophia Adiposo Genitalis, Retinitis Punctata Albescens, Retinitis Pigmentosa, and Endocrine Cataract, Ann. ocul. **185**:543, 1952.

the retinal vessels were narrowed, the optic disk was pale, and throughout the fundus were numerous white and brilliant points typical of retinitis punctata albescens. Visual acuity was 0.5 in the right and left eyes, and the visual fields were contracted. By 1951 a posterior polar cataract had developed; the vessels were extremely narrowed, and typical deposits of osteoblast-shaped pigment were seen over both fundi, especially in the periphery. Visual acuity in the right eye was limited to light perception, and the left eye was blind. The author cites this case as one of retinitis punctata albescens which changed into true retinitis pigmentosa. This indicates that the two diseases not only form one group but may occur in the same patient.

The effect of block of the cervical sympathetic chain with 1% cocaine hydrochloride was tested in 14 patients with retinitis pigmentosa. Seven of these showed subjective improvement in vision, but there was no definite improvement in visual fields or visual acuity. When nine of the same patients were given injections of saline in the region of the superior cervical sympathetic ganglion, six of them described subjective improvement; but, again, there was no change in visual fields or visual acuity. The authors, Kennedy and McGannon,[148] conclude that any benefit derived from cervical sympathetic block is purely psychogenic.

Renard and Mallet[149] discuss the problem of retinal vascular spasm in retinitis pigmentosa and conclude that vascular changes are secondary to an organic disturbance which causes retinitis pigmentosa. They report the case of a man aged 25 who did not show improvement in spite of various attempts to produce vasodilatation with nicotinic acid, benzazoline, stellate ganglion block, and denervation of the carotid sinus. There was temporary improvement in vision following section of the right superficial nerve plexus, but after this the disease progressed as before.

Feist and Prskavec[150] discuss the treatment of retinitis pigmentosa with placental therapy and state that this produces vasodilatation and a hormone-type of action.

Straub and Heberling[151] report five cases of the Laurence-Moon-Bardet-Biedl syndrome in three families. In the first family two girls and one boy, aged respectively 14, 7, and 6 years, had ectopic pupils and the usual manifestations of the disease. In the second family, a woman aged 24 with a picture compatible with retinitis pigmentosa sine pigmentosa had development of true retinitis pigmentosa.

Friede[152] discusses the use of benzazoline, milk, aloe, and hormone injections; paracentesis; short-wave diathermy; oxygen inhalation, and vitamins in the treatment of retinitis pigmentosa. Some improvement was thought to have been obtained in 5 of the 39 cases.

148. Kennedy, R. J., and McGannon, W. J.: Stellate-Ganglion Block in Retinitis Pigmentosa, A. M. A. Arch Ophth. **47**:287, 1952.

149. Renard, G., and Mallet, R.: Arterial Spasm in Retinitis Pigmentosa, Bull. Soc. opht. France, 1952, p. 589.

150. Feist, S., and Prskavec, F.: Mode of Action of Placental Therapy in Retinitis Pigmentosa, Klin. Monatsbl. Augenh. **121**:329, 1952.

151. Straub, W., and Heberling, V.: Addition to the Symptomatology and Clinical Findings of the Syndrome of Laurence-Moon-Bardet-Biedl, Klin. Monatsbl. Augenh. **120**:289, 1952.

152. Friede, R.: Therapy of Congenital Retinitis Pigmentosa, Klin. Monatsbl. Augenh. **120**:605, 1952.

Rama,[153] after a study of the clinical findings and history of a number of patients with retinitis pigmentosa, concludes that the clinical picture was determined by various conditions beside hereditary factors. He treated *27* patients with pigmentary degeneration of the retina by injection and instillation of melanophore hormone. The general effect was good on visual acuity, slight on visual fields, and almost nil on the light sense. These results are substantially the same as those obtained by other authors with other forms of treatment.

Ashton and Sorsby[154] describe the pathological findings in the eyes of two affected members of a family with a peculiar type of fundus dystrophy. This condition had previously been described by Sorsby in 1949. It starts at about the age of 40 with a central retinal hemorrhage, edema, and exudates. During the intermediate and progressive stages, exposure and sclerosis of the choroidal vessels occur, with associated pigment proliferation. In the final stage of the disease, some 30 years after the onset, widespread choroidal atrophy occurs.

Histological examination reveals some atrophy of the choriocapillaris, but this is not abnormal considering the age of the patient. Numerous ruptures are found in Bruch's membrane. Adjacent to these ruptures newly formed tissue separates Bruch's membrane from the retina. These masses are composed of fibroblasts from the choroid, glial tissue from the retina, and reticular substance from the pigment epithelium. The pigment epithelium is disorganized and collected around the retinal vessels. The outer layers of the retina are relaced with glial tissue. The authors conclude that these findings are similar to those which have previously been reported in cases of angioid streaks and disciform degeneration of the macula.

The case of a woman aged 47 with unilateral pigmentary changes in the fundus and a vitreous cyst is described by Wagenaar.[155] When the patient was first seen, in 1947, vision in the right eye was 5/15, and she had a very slight loss of visual field. Visual acuity and the visual field in the left eye were normal. By 1947 the patient's visual field was constricted to approximately 10 degrees. Her visual acuity was reduced to 5/30. The left eye remained normal. The author states that two similar cases had been reported previously.

François and Verriest[156] reviewed the literature on unilateral retinitis pigmentosa and found 56 reports. In most of the cases described there was no history of consanguinity or heredity. They report an additional case, that of a woman aged 43 who had a macular coloboma in the right eye that was not due to toxoplasmosis. This possibility was ruled out by a negative reaction to the Sabin dye test and the absence of cerebral calcifications. Visual acuity was 5/15. The rest of the ophthalmoscopic examination was noncontributory. There were no signs of retinitis pigmentosa in the right eye. However, the left eye presented a typical picture of retinitis pigmentosa, with contraction of the visual field to 10 degrees, defective dark adaptation, and absence of an electroretinogram. The patient's visual difficulty in the left eye began when she was 21 years of age.

153. Rama, G.: Etiology, Prognosis, and Treatment of Pigmentary Degeneration of the Retina, with Particular Reference to Therapy with the Melanophore Hormone, Ross. ital ottal. **24**:143, 1952.

154. Ashton, N., and Sorsby, A.: Fundus Dystrophy with Unusual Features: A Histological Study, Brit. J. Ophth. **35**:751, 1951.

155. Wagenaar, J. W.: Vitreous Cyst with Retinitis Pigmentosa: A New Syndrome? Brit. J. Ophth. **36**:492, 1952.

156. François, J., and Verriest, G.: Unilateral Pigmentary Retinopathy, Ophthalmologica **124**:5, 1952.

Lijó Pavia[157] describes a rare type of tapetoretinal degeneration with a luminescent crystalline appearance of the macular area which has not been previously recorded. The author studied four cases. He thinks that the luminescent crystalline points are probably cholesterol crystals in the deeper layers of the retina. Visual acuity is seriously diminished. No familial history of this type of ocular difficulty was obtained in any case, nor was there a history of previous ocular inflammation. Hypercholesteremia was present in all cases. Retinal photographs of the lesion are included.

A family in which three members were found to have central retinal degeneration of Stargardt's type is described by Klimková-Deutschová and Velický.[158] The macular lesions, which were oval in the horizontal diameter, consisted of a fine, stippled pigmentation with sharply defined, yellowish spots between them. The peripheral fields and dark adaptation were normal. In the patient with the most typical macular degeneration, there was associated obesity, with maximal fat distribution over the hips and abdomen, a small, flat sella turcica, acromicria, mental retardation, and lesions of the nervous system. Endocrine cataract was present in both lenses.

Bruna[159] describes two cases of Stargardt's type of macular degeneration.

Schubert and Bornschein[160] produced retinal damage in rabbits by injecting iodoacetate intravenously. The electroretinogram was extinguished 22 days after injection. Histological examination showed selective destruction of the rods and cones.

Fornaro[161] notes that the administration of melanophore hormone inhibited the development in rabbits of pigmentary degeneration in the retina which had been experimentally induced by sodium iodate. Corticotropin is incapable of impeding its onset but lessens the severity of the manifestations.

Sorsby[162] and his co-workers discuss in some detail the clinical and genetic aspects of choroideremia. They conclude that there is no adequate evidence from their studies or from reports in the literature that there is any specific relation between retinitis pigmentosa and choroideremia.

MACULAR LESIONS

A review of the literature and a report of one case of central angiospastic retinopathy is given by Buxeda.[163] The patient was a white man aged 36 whose visual acuity was reduced to 20/40+3 in the right eye and 20/20 in the left eye.

157. Lijó Pavia, J.: Rare Macular Tapetoretinal Degeneration of Luminescent Crystalline Aspect: Three First Observations, Rev. oto-neuro-oftal. **27**:27, 1952; II. ibid. **27**:51, 1952.

158. Klimková-Deutschová, E., and Velický, J.: Stargardt's Degeneration of the Retina with Neurological Complications, Ophthalmologica **123**:162, 1952.

159. Bruna, F.: An Unusual Form of Tapetoretinal Degeneration in the Macular Area, Boll. ocul. **30**:569, 1951.

160. Schubert, G., and Bornschein, H.: Specific Damage of Retinal Elements Caused by Iodoacetate, Experientia **7**:461, 1951.

161. Fornaro, L.: Action of ACTH and of the Melanophore Hormone on Experimental Pigmentary Retinal Degeneration of the Rabbit, Gior. ital. oftal. **5**:148, 1952.

162. Sorsby, A.; Franceschetti, A.; Joseph, R., and Davey, J. B.: Choroideremia: Clinical and Genetic Aspects, Brit. J. Ophth. **36**:547, 1952.

163. Buxeda, R.: Central Angiospastic Retinopathy: Review of the Literature and Report of One Case, Am. J. Ophth. **35**:1769, 1952.

Ophthalmoscopic examination showed a slight pallor of the macular area in the right eye, with absence of the foveal reflex. There were also a few small yellowish-white spots in the perifoveal area. Visual acuity returned to 20/20–1 in one week.

Nicholls [164] reports 23 cases of central retinal disease related to a disturbance of the retinal and choroidal circulations. Among the diagnoses in these cases were central choroidal sclerosis, senile macular degeneration, hypertensive arteriolar sclerosis, and central angiospastic retinopathy. The author states that, although these conditions are traditionally considered as separate entities, actually they merge one into the other. Some improvement in these conditions is reported to have followed the administration of 50 mg. of nicotinic acid by mouth three times a day.

Rieger [165] reports 10 cases of central serous retinitis and discusses the possibility that these lesions are due to acquired toxoplasmosis in adults.

The case of a woman aged 40 who had chorioretinitis centralis serosa is described by Riehm.[166] This case was remarkable in that there was a horizontal line of yellow exudate beneath the retina which moved with a change in the position of the patient's head. The lesion subsided after one and one-half years.

François and Lambrechts [167] state that retinal cysts may be of four types: (1) cystoid degeneration of the retina, (2) secondary retinal cysts, (3) congenital retinal cysts, and (4) parasitic cysts. They describe two cases of retinal cyst, one in a man aged 28, who presented an acquired cyst of the macular area. When he was first seen, vision in the right eye was 0.8. Ten months later his visual acuity dropped to 0.4. Three small hemorrhages developed in the macular area. The other case was that of a woman aged 54 who had cysts in the extreme temporal periphery in both eyes. No change occurred in these cysts in 12 years of observation.

Palich-Szántó [168] describes two cases in which a blue-yellow cyst of approximately 4 D. elevation occurred in the macular area. Only one eye was involved in each case. Both cysts were transparent, with sharp borders surrounded by some pigment spots. A diagnosis of congenital retinal cyst was made. No other abnormalities were noted in the eye. The literature on this subject is reviewed.

A case of a 9-year-old boy who had a congenital prepapillary cyst that extended into the retina is described by Tower.[169] On ophthalmoscopic examination the right fundus showed a grayish ovoid mass, raised about 4 D. above the level of the papilla. The disk was hidden by the growth, which extended far into the retina. On the nasal border of the disk the remnants of Cloquet's canal was seen. With movements of the eye the cystic growth showed a slight shimmering motion, and the proximal portion of the superior temporal vessel, otherwise hidden from view, was momentarily exposed. Visual acuity in this eye was limited to counting fingers. The left fundus was normal, and visual acuity was 20/20.

164. Nicholls, J. V. V.: On the Character and Management of Circulatory Disturbances of the Central Retina, Am. J. Ophth. **35**:1753, 1952.

165. Rieger, H.: Further Cases of Retinitis Exsudativa Externa Retinae Centralis Suspected of Acquired Adult Toxoplasmosis, Klin. Monatsbl. Augenh. **121**:33, 1952.

166. Riehm, W.: Horizontal Borderline Caused by Homogeneous Yellow Exudate Behind the Macula in Chorioretinitis Centralis Serosa, Klin. Monatsbl. Augenh. **121**:76, 1952.

167. François, J., and Lambrechts, J.: Retinal Cyst, Ann. ocul. **185**:348, 1952.

168. Palich-Szántó, O.: Retinal Cysts, Ophthalmologica **122**:283, 1951.

169. Tower, P.: Congenital Prepapillary Cyst, A. M. A. Arch. Ophth. **48**:433, 1952.

Hermann [170] thinks that the juvenile type of Stargardt's macular degeneration, the adult type of Best, and familial forms of limited and symmetrical macular lesions should be classified under the same heading. He suggests that an appropriate name for these lesions is central tapetoretinal degeneration. In these conditions the fundus lesions are characterized by a yellowish or slightly granular circular lesion in the macular area of approximately 1 disk diameter or less. The ophthalmoscopic appearance of the lesion would suggest that the patient's visual acuity should be markedly reduced. However, vision is generally amazingly good. There are usually a slight progression of the macular changes and deterioration of central visual acuity over a long number of years. The author thinks the lesion can best be accounted for by an atrophic, tapetum-like lesion, rather than a change in the retina itself. Macular changes of the type just described were observed in three generations of a family. The members affected were a grandmother, whose lesion began at the age of 40; a daughter, whose lesion began at 25, and a granddaughter, the onset of whose disease was at 6 years of age. Excellent colored drawings illustrate the macular lesions in these three patients.

Blankstein and Feiman [171] describe four patients with pigmentary changes in the macular area whose mothers had rubella during the first trimester of pregnancy. The visual acuity in all but one of the six eyes of these patients was 20/30 or better.

Biro [172] describes the ophthalmic conditions which are caused by consanguinity. Macular lesions of a granular type with loss of central visual acuity were noted in five patients.

Amsler [173] states that metamorphopsia may be produced by myopia, senile macular degeneration, central chorioretinitis and central serous retinititis, cystic macular degeneration, detachment of the retina, and phototraumatism. The best method of examining this type of lesion is the use of squares the author has designed, in which the patient fixes on a central point and a scotoma is indicated by a deformity of the squares.

François and Deweer [174] present observations on four families which confirm their opinion that senile macular dystrophy is a hereditary disease that should be classified as central hereditary tapetoretinal degeneration.

Stargardt's macular degeneration in four of five children in one family is described by Bartolozzi.[175] Three of the members, aged 17, 16, and 11, respectively, had well-defined macular lesions with central-field changes. A girl, aged 12 years, had a normal fundus, but a relative central scotoma could be demonstrated on a Bjerrum screen. The youngest child, aged 7 years, had no fundus changes but was too young for accurate field studies.

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170. Hermann, P.: Central Tapetoretinal Degeneration, Arch. opht. **12**:188, 1952.

171. Blankstein, S. S., and Feiman, L. H.: Macular Pigmentation Following Maternal Rubella, Am. J. Ophth. **35**:408, 1952.

172. Biro, I.: Ophthalmic Conditions in Consanguinity, Klin. Monatsbl. Augenh. **119**:585, 1951.

173. Amsler, M.: Contributions to the Study of Retinal Metamorphopsia, Thesis, Zurich, 1951.

174. François, J., and Deweer, J. P.: Senile Hereditary Degeneration of the Macula, Ann. ocul. **185**:136, 1952.

175. Bartolozzi, R.: Familial Macular Heredodegeneration, Arch. Soc. oftal. hispano-am. **12**:163, 1952.

RETINAL DETACHMENT

A comprehensive and very instructive review of retinal detachment was presented at a symposium held at the 56th annual session of the American Academy of Ophthalmology and Otolaryngology. Post [176] gives a definition of terms. He also states that, in the opinion of the panel, breaks in the continuity of the retina are the most essential predisposing factor in the production of retinal detachment. He reports that such breaks may be caused by a localized atrophying process in the retina, a localized pull on the vitreous, or trauma.

Wadsworth [177] discusses the pathological changes of retinal detachment under three phases: anatomical changes, sequence of events, and microscopic changes following treatment. Each phase is illustrated with excellent photomicrographs. He demonstrated a detachment of the retina which had resulted from a break near the ora serrata following a contraction of the vitreous. An adherent vitreous strand was still attached to the operculum. Fluid had seeped beneath the retina, and cystic changes had occurred in the outer layers of the retina.

He discusses the reason for loss of retinal function in long-standing retinal detachment and attributes it to (1) cystic changes in the plexiform layers, (2) a decrease in the nuclear layers and fusion of one layer to another, (3) disappearance of the ganglion cells, and (4) degeneration of the rods and cones.

In some instances, the prognosis for reattachment is reduced because of (1) contraction of the glial supporting tissue, with resultant shortening of the retina, and (2) proliferation of tissue on the surface of the retina, which causes retinal folds. This new-formed tissue on the surface of the retina may be derived from the nonpigmented epithelial cells of the ciliary body or proliferation and contraction of the cells normally present on the inner surface of the retina.

In some instances a detachment will remain localized in one area. The outer limits of the detachment are characterized clinically by grayish or pigmented lines. Histologically, these are represented by chorioretinal adhesions, which result from a proliferation of the pigmented epithelium of the retina. Three things may happen to these friction lines: 1. The adhesion may be sufficiently dense to localize the area of detachment. 2. The subretinal fluid may exert sufficient force to break the chorioretinal adhesion, causing a further extension of the detachment. 3. The retina may tear at the point of the adhesion, thereby forming a second break and complete detachment.

A ruptured cyst in the periphery of the retina or in the macular area does not always result in retinal detachment, for two reasons: 1. In some instances only the inner layers of the retina break, leaving the outer layers intact. 2. In other instances chorioretinal adhesions occur around the periphery of the break, which prevent a seepage of the fluid beneath the retina.

Histological examination of eyes that have been treated by diathermy for retinal detachment reveals that diathermy applications to the sclera result in shrinkage and flattening of the sclera, areas of choroiditis, and, in successful cases, chorio-

176. Post, L. T.: Symposium: Retinal Detachment: Introduction and Definition of Terms, Tr. Am. Acad. Ophth. **56**:369, 1952.

177. Wadsworth, J. A. C.: Symposium: Retinal Detachment: Etiology and Pathology, Tr. Am. Acad. Ophth. **56**:370, 1952.

retinal adhesions. If the areas of chorioretinal adhesions are properly placed, they will seal the retinal breaks and form a new ora serrata. If the operation is to be successful, it is essential that all breaks in the retina be sealed.

The author thinks that in perforating diathermy chorioretinal adhesions result from proliferation of the pigment epithelium rather than from an ingrowth of episcleral tissue in the area of perforation.

Schepens [178] discusses the diagnostic and prognostic significance of factors found in preoperative examination. The earliest symptoms of retinal detachment are localized flashes of light, a sudden increase in vitreous floaters, and, frequently vitreous hemorrhages. When the detachment extends, a gray cloud appears in the visual field and light flashes disappear. Between 40 and 60% of all detachments occur in patients with myopia, and of the patients seen by the author over 20% were aphakic. Signs of anterior uveitis are fairly frequent in patients with retinal detachment and do not affect the prognosis. There may also be atrophy of the posterior pigment layer of the iris, which indicates marked degeneration of the extreme periphery of the fundus.

In most cases of idiopathic detachment the vitreous is degenerated and detached, but vitreous detachment has little effect on the operative prognosis and frequently is found in patients without retinal detachment. Therefore, the value of vitreous detachment as a prognostic or precursory sign of retinal detachment is difficult to evaluate. This is not true, however, of vitreous strands, as will be described later.

In nearly every case of retinal detachment a field defect may be detected, provided one uses small test objects or reduced illumination. The one exception to this is the eyes with a small flat detachment in the far periphery of the fundus. Often in central flat detachments only a relative defect is found.

The extent and location of the detachment often indicate the location of the retinal breaks. If only one upper quadrant is affected, breaks are probably present in the superior part of the detachment. If an inferior detachment extends considerably above the horizontal meridian, there is nearly always a break in the upper part of the retina. If a break in the upper quadrant is present for over a month, the subretinal fluid will often sink to the lower half of the fundus. It is generally thought that total detachment has a poor prognosis. However, cure often results if all the retinal breaks are detected and repaired.

Most retinal breaks are found either in the equatorial region or in the ora serrata. Equatorial breaks are usually located in the upper temporal quadrant, where either holes or tears occur in degenerated areas.

Breaks near the ora serrata cause a detachment, which, as a rule, progresses slowly. They are usually one of the following types: 1. Small and multiple, forming tiny dialyses or breaks related to meridional folds. This type of break is usually found in patients over 45 years of age and may be associated with quiescent peripheral uveitis, posterior cortical cataract, or deep-chamber glaucoma. This type of break also occurs in aphakic eyes in which a retinal detachment develops. 2. Single dialysis. This type occurs in young patients predominantly, affects the lower temporal quadrant, and has a tendency to bilaterality. 3. Multiple

178. Schepens, C. L.: Symposium: Retinal Detachment: Diagnosis and Prognostic Factors as Found in Preoperative Examination, Tr. Am. Acad. Ophth. **56**:398, 1952.

traumatic dialyses. These breaks are often impossible to see unless the sclera is strongly indented for examination. They are generally located either above and nasally, or below and temporally.

The most valuable method of detecting small peripheral retinal detachments or breaks in the retina near the ora serrata is the use of Schepens' indirect ophthalmoscope and scleral indentation.

If the macula is detached, normal visual acuity is seldom recovered. The use of slit-lamp illumination and stereopsis will help in differentiating between a macular cyst and a true macular break.

The presence of vitreous-retinal adhesions, which indicates a very poor prognosis, can usually be detected by the presence of three types of fundus lesions. The first is characterized by fixed folds, which may be circular and located near the equator generally, or star-shaped and located either below the horizontal meridian or near the macula. The second is a massive retraction of the vitreous, in which all the retinal folds are fixed, with most of them radiating from the disk. This type occurs most frequently in long-standing detachments following vitreous hemorrhage of moderate or large amount, and after retinoplexy. The third fundus change that indicates a very poor prognosis is the presence of equatorial breaks with rolled-up, convex edges.

A true retinal detachment can be differentiated from a retinocele or a detachment associated with a choroidal tumor by the following features: A retinocele appears as a limited elevation of smooth, thin retina showing obliterated vessels, glittering spots resembling snowflakes, and large reflections, which are sometimes visible through an undilated pupil. The resulting field defect has steep limits; usually no retinal break is present, and the lesion is often stationary. A choroidal tumor is not accompanied by retinal breaks, and a gray lesion with mottled pigment, and occasionally small hemorrhages, can be seen beneath the retinal detachment.

The various types of retinal breaks and retinal detachments described in Schepen's paper are excellently illustrated by colored photographs.

Pischel[179] describes the treatment of retinal detachment. He again emphasizes the importance of localization and accurate recording of the location of retinal breaks prior to operation. He states that five requirements must be met in any operation that cures retinal detachment by closing the breaks: 1. Exudative chorioretinitis must be produced. 2. It must be produced in the proper place. 3. The retina must be brought into contact with the choroiditis so that the edge of the hole or tear will touch the exudate. 4. It must be allowed to remain in firm contact until adhesions between the choroid and the retina are established. 5. The adhesions must be watertight.

Pischel's detailed description of the operative procedure does not lend itself to abstracting and should therefore be read in its entirety. Some points stressed are frequent observation of the fundus during operation and accurate localization of retinal breaks. The simplest, safest, and easiest method of regulating retinal coagulation is by use of a diathermy current. His choice of instruments for coagulation is

179. Pischel, D. K.: Symposium: Retinal Detachment: Treatment of Retinal Detachments; Localization of Breaks, Description of Typical Operative Procedure, Tr. Am. Acad. Ophth. **56**:419, 1952.

the Walker diathermy unit, partial penetrating electrodes, and small, penetrating pins. The greater part of the coagulation is done with partial penetrating electrodes (0.5 or 1 mm. in length). The perforating pins are used to localize the retinal breaks and to surround the area of the break when the retina is markedly elevated. Eight to 12 pins are introduced to produce drainage points for the subretinal fluid. He condemns the use of scleral trephines, which may allow a sudden gush of subretinal fluid, for in many instances the retina is included in the openings and folds in the retina will be produced which will doom the operation to failure.

When the retina is extremely elevated at the end of operation, saline or air is injected into the vitreous cavity through the pars plana in an attempt to push the retina back on the coagulation points. If the eye is aphakic, the injections may be done through the cornea into the anterior chamber.

Kronfeld[180] divides his discussion of the postoperative care and complications into three categories: (1) the routine postoperative course, (2) irregularities in the postoperative course, and (3) complications. He thinks that the postoperative position of the patient should be a sensible compromise between placing the retinal breaks in the lowest position and the ability of the patient to maintain such a position. Avoidance of eye movements is more important than the position of the patient's head. The fundus is not examined until the sixth or seventh postoperative day. Movements of the eye which occur during examination may disrupt the chorioretinal adhesions, for they are not firmly attached before the end of the first week. The patient is kept in bed for approximately two weeks after operation and is required to wear pinhole glasses for about eight weeks from the day of operation. Pinholes that are 2 to 3 mm. in diameter are as effective as, and much more practical than, those of 1 mm. diameter.

Irregularities in the postoperative course are (1) incomplete settling down of the retina, (2) complete settling down of the retina followed by recurrence, (3) complete settling down followed by a detachment in a new area, (4) no visible response to operation, and (5) progression of the detachment.

The commonest cause of incomplete regression is a slight leak or gap in the barrier, which causes a small residual detachment. If after a month of observation there is no evidence on ophthalmoscopic examination of closure of the gap, a second operation is indicated. The prognosis is usually good, since the non-spreading of the detachment indicates that it is fundamentally benign.

When a detachment recurs in the sector originally involved and surgically treated after an apparently successful result, the prognosis is poor. New breaks just posterior to the surgical chorioretinal adhesions or an actual tearing away of the retina from these adhesions usually proves to be the cause of the recurrence. The appearance of the retina in several such cases observed by the author has suggested an intrinsic shrinkage or retraction of the retina. This is possibly due to injurious effects of unnecessarily strong and extensive coagulation.

Another typical variation in the postoperative course is complete subsidence of the original detachment, followed one to three months later by a new detachment in a new location. This is usually caused by atrophic spots in another area

180. Kronfeld, P. C.: Symposium: Retinal Detachment: Postoperative Care and Complications, Tr. Am. Acad. Ophth. **56**:432, 1952.

of the fundus, which turn into breaks and cause a new detachment. This is probably not related to the first operation, and a second operation aiming at the new breaks has a good chance of being successful.

In some new detachments, especially if the new site is diametrically opposite the original one, the mechanism seems to be different, and a relationship to some event of the operation is very likely. Often this is due to a contraction of vitreous bands which are attached to the area of the original operation and to the retina in the area of the new detachment. When patients with this type of detachment are immobilized, the retinal detachment does not settle back, and the prognosis is poor.

When the position of the retina is not visibly affected by the operation, the cause may be (1) vitreous strands or bands holding the retina, (2) internal retinal rigidity due to glial proliferation, or (3) inadequate drainage. In the first two instances the prognosis is extremely poor. In the last adequate drainage may effect a cure if the coagulations are approximately one week old.

The late form of progression of detachment occurs characteristically three to six weeks after an apparently unsuccessful and uncomplicated operation. The retina, almost overnight, assumes an ominous umbrella shape. Massive retraction of the vitreous appears to be the underlying mechanism of this complication.

True complications which occur during the postoperative period are a benign form of iridocyclitis which follows extensive application of the diathermy current to any, but especially to the anterior, portion of the sclera, suppurative infections, and hemorrhages into the vitreous and choroid.

Hughes[181] made a thorough statistical analysis of 375 consecutive cases of primary retinal detachment in both phakic and aphakic eyes operated on by members of the symposium, the attending staff, or residents in their respective hospitals. Each patient had been followed for at least six months since the last operation. Although the proportion of successful operations was lower in aphakic than in phakic eyes (44 and 72% respectively), the committee was of the opinion, borne out by the analysis, that similar factors predisposing to success or failure were present in the two groups, without its being assumed that the presence or absence of the lens per se had any influence on the result of operation. Of the two groups considered together, 67% were cured by operation.

The factors which adversely affected the prognosis for reattachment of the retina included (1) myopia; (2) peripheral cystic degeneration; (3) size of the detachment, although 48% of the total detachments were cured; (4) failure to detect and localize retinal breaks; (5) failure of the retina to fall back in apposition to the choroid; (6) blind, and possibly excessive, barrage with diathermy current, and (7) sclerotomy and sclerectomy for the evacuation of subretinal fluid.

The relatively poorer prognosis in aphakic than in phakic eyes may be partially explained by their larger detachments, less frequent localization of the breaks, and less flattening out on bed rest.

The results of 38 scleral resections, performed largely on aphakic eyes or after failure of diathermy, showed 18% cures.

181. Hughes, W. F., Jr.: Symposium: Retinal Detachment: Evaluation of Results of Retinal Detachment Surgery, Tr. Am. Acad. Ophth. **56**:439, 1952.

Visual improvement is often dramatic after surgical reattachment of the retina, even though the macula has previously been detached.

At the same meeting of the American Academy of Ophthalmology and Otolaryngology, Arruga [182] delivered the address of the guest of honor. His paper concerned the surgical treatment of retinal detachment and may be divided into two parts: (1) a brief discussion of some of the fundamental considerations in the surgical treatment of retinal detachment, and (2) the presentation of 34 colored illustrations of various types of retinal breaks and detachments. A brief description of these included the type of treatment indicated and the percentage of cures to be expected.

He stressed the fact that many failures are due either to defective localization of tears or to an excessive diathermy action.

Simpson [183] describes a syndrome which he thinks is due to the appearance of a piece of retina in the vitreous. The symptoms consist of the sudden appearance of a black floater in the line of vision, which is frequently accompanied by slight generalized haziness of vision. On ophthalmoscopic examination a single, black floater can be seen. This is more or less solid in appearance though sometimes mesh-like; irregular in shape, and fringed with vitreous opacities. In almost every instance, especially if the solid opacity is of fair size, a shadow is cast upon the retina. In three cases of vitreous opacities of this type, holes were found in the peripheral retina without retinal detachment. The author theorizes that in other cases in which holes cannot be found the piece of retina originates from the far periphery, beyond the limits of visibility with the ordinary ophthalmoscope examination.

Schepens [184] defines a subclinical retinal detachment as one in which a flat separation of the retina is present in the far periphery that will escape detection unless careful, methodical ophthalmoscopic examination is done. The visual fields and central visual acuity in such cases are usually not affected by the detachment.

When careful, painstaking examination is made with the direct ophthalmoscope, many subclinical detachments may be detected. In three types of cases, however, a diagnosis must be made with other methods: (1) cases in which a subclinical detachment is hidden by a fine vitreous haze in the periphery of the fundus, (2) cases in which a subclinical detachment is so peripheral that it cannot be seen with the direct ophthalmoscope, and (3) cases in which the nature of the detachment is such that diagnosis cannot be made without the use of the indirect ophthalmoscope and scleral indentation.

Subclinical detachments may be divided into two types: (1) that in which there is no visible retinal break, but only a small, extremely peripheral detachment, and (2) that in which a retinal break is present but the detachment is so small that it is difficult to detect with the ophthalmoscope. Schepens' presentation was exclusively concerned with subclinical detachments in which a retinal break could be found. He studied 110 affected eyes in 102 patients. Only 43 of the patients complained of one or more of the following symptoms: blurred vision, floating

182. Arruga, H.: Certain Considerations of Surgical Treatment of Retinal Detachment, Tr. Am. Acad. Ophth. **56**:535, 1952.

183. Simpson, G. V.: Pieces of Retina in the Vitreous, Tr. Am. Ophth. Soc. **49**:75, 1951.

184. Schepens, C. L.: Subclinical Retinal Detachment, A. M. A. Arch. Ophth. **47**:593, 1952.

specks, and flashes of light in one quadrant. In one-half the patients there was a single retinal break, and the other half had multiple breaks. Retinal holes were much more frequent than tears. The majority of the holes were in the region of the ora serrata, whereas the tears were commoner near the equator. In about one-quarter of the eyes with breaks near the ora serrata, the breaks were related to meridional folds, which were found more frequently in the upper half of the fundus and affected the upper nasal quadrant four times as often as the lower temporal quadrant. Visible chorioretinal degeneration was found in two-thirds of the eyes and was usually, although not always, located in the same area as the retinal break.

Two relatively important syndromes were found in patients with subclinical detachments. The first was observed in four female patients and was characterized by macular degeneration, generally bilateral, and advanced peripheral degeneration. Retinal breaks were found in the degenerated areas. Recognition of this syndrome is important in cases of clinical detachment because of prognosis for visual acuity is poor. The second syndrome is important in cases of clinical detachment because the prognosis for visual acuity is usually poor. The second syndrome occurred usually in cases of subclinical detachment with alterations of the fundus that were near the ora serrata. The retinal breaks were usually related to meridional folds. Other characteristics were opacities in the posterior cortex of the lens; peripheral uveitis of the disseminated type, generally without signs of anterior uveitis, and deep-chamber glaucoma.

Thirty-nine (or 35%) of all eyes with subclinical detachments were operated on. Very weak applications of surface diathermy alone were used in most cases. Perforations were used only when it was thought that enough subretinal fluid was present to justify it. Reattachment followed surgical obliteration of the breaks in 38 eyes. One patient had what appeared to be a good result for three months, when a clinical detachment appeared, with a new break in the upper temporal quadrant on the disk side of the barrage. A second operation, with surface and perforating diathermy, resulted in a massive vitreous reaction and complete retinal detachment.

The subsequent course of the 79 eyes with subclinical detachments which were not operated on is not specifically mentioned, but it is noted that clinical detachments developed in "several cases."

Bonavolonta[185] observed 300 eyes with retinal detachments. One hundred ninety-seven (more than 65%) of these showed chorioretinal disease. In 80 eyes (26%) the lesions were located in the equatorial region; 42 (14%) were disseminated in type; 6 (2%) showed a single focus, and 63 (21%) were mixed types of chorioretinitis. In 65 eyes (21%) the retinal tear corresponded to the chorioretinal lesions.

LaRocca[186] described a patient with complete retinal detachment in an aphakic eye. The detachment had occurred a few days after the institution of diisopropyl fluorophosphate therapy for secondary glaucoma following cataract extraction.

185. Bonavolonta, G.: Importance of Chorioretinitis and Retinal Tears in Idiopathic Retinal Detachment, Boll. ocul. **31**:206, 1952.

186. LaRocca, V.: Retinal Detachment from Di-Isopropyl Fluorophosphate in an Aphakic Eye, New York J. Med. **52**:1329, 1952.

Krümmel [187] described the topography of tears in cases of idiopathic retinal detachment. Of 523 patients, 60% had the tears in the right eye and 40% in the left eye. The tears occurred mainly in the upper temporal quadrant, usually near the insertion of the superior oblique tendon. Tears occurred in the upper temporal quadrant of the right eye in 66.2% and of the left eye in 57.2%. The placement of the tears suggested that the mechanical action of the superior oblique produces a swinging action of the vitreous in sudden movements of gaze. This results in cystoid changes in the region of the insertion of the superior oblique and, finally, in tears in the retina in this area.

Coles and Laval [188] report that eyes operated on for cataract associated with neurodermatitis (atopic dermatitis) have a poor prognosis. They cite one case as an illustration and present a statistical study of 63 eyes of this type on which surgery had been done. Blindness resulted in 23 (36%) of these eyes. The retinal complications were retinal bleeding, edema, and retinal detachment.

Levy [189] presented the pedigrees of two families showing retinal detachment consistent with x-chromosomal inheritance. In one family seven males, in three generations, and in the other two males, in two generations, were affected. The nature of the detachments varied in the first family. In the younger affected members detachments were present in the lower portion of the retina, and at least one of these appeared to be related to a retinal cyst. This family also had varying degrees of macular degeneration, vitreous veils, and dendritic lines in the retina. The detachments in the second family were of the commoner variety.

Juler [190] reports a follow-up study on two children with cystic disease of the retina. The cases were originally described in 1947. These children were not related, and a familial history of retinal detachment is not mentioned. However, the author relates these detachments to a familial inheritance. In both these patients the area of retinal involvement has remained unchanged for eight years. The nonprogressive nature of the lesions suggests that treatment should be nonoperative.

Braley and Allen [191] describe a corneal contact prism and corneal contact lens for examination of the peripheral and central fundus with the slit-lamp biomicroscope. The instrument for examining the periphery consists of a plastic planoconcave prism, a spring carriage, and a plastic speculum. The same three components are used to examine the central fundus except that a planoconcave lens replaces the prism.

187. Krümmel, H.: Topography of Tears in Idiopathic Retinal Detachment, Klin. Monatsbl. Augenh. **119**:388, 1951.

188. Coles, R. S., and Laval, J.: Retinal Detachments Occurring in Cataract Associated with Neurodermatitis, A. M. A. Arch. Ophth. **48**:30, 1952.

189. Levy, J.: Inherited Retinal Detachments, Brit. J. Ophth. **36**:626, 1952.

190. Juler, F.: Further Report on Cystic Diseases of the Retina in Children, Tr. Ophth. Soc. U. Kingdom **71**:199, 1951.

191. Braley, A. E., and Allen, L.: A Corneal Contact Prism and a Corneal Contact Lens for Slit Lamp Biomicroscopic Examination of the Peripheral and Central Fundus, Tr. Am. Ophth. Soc. **49**:477, 1951.

Gonzales [192] advocates the use of the Goldman contact lens and biomicroscope for examination of tears in the periphery of the fundus. He has devised a special double mirror which directs the slit-lamp beam horizontally. He suggests that a syrupy, transparent fluid, isotonic with tears, be placed under the contact lens so that air bubbles between the lens and cornea are avoided.

Dixon [193] describes two binocular indirect ophthalmoscopes. The first consists of a Bausch and Lomb binocular magnifier attached to a head band with a swivel. The light source is the National Headlight Illuminating Unit with a condenser, which can be regulated to furnish as much light as desired. The second unit is exactly the same except that the rhomboid prisms are replaced by four front-surfaced mirrors.

Smith [194] describes a unique technique for pushing the retina into contact with the diathermy punctures when spontaneous flattening of the retina does not take place at the end of operation. Routine diathermy punctures are made in the area of the detachment, and as much subretinal fluid as possible is removed by suction. An incision is then made through the sclera, 5 mm. from the limbus, on the side of the globe opposite the diathermy punctures; 1.5 cc. of air is then injected into the suprachoroidal space. This results in a choroidal detachment which pushes the subretinal fluid out through the diathermy punctures. Five patients, all of whom had a fairly poor prognosis, were operated on in this manner. Reattachment occurred in three patients, and two had a recurrence of the detachment. Of the three patients with reattachments, one had a vitreous hemorrhage, but four months after operation the retina was flat and visual acuity was 6/36.

Three patients with retinal detachments in the superior part of the fundus were operated on by Jervey [195] and allowed early ambulation. One of the patients was never placed in bed, and the other two were allowed to be up on the third postoperative day. These two patients had recurrences of their retinal detachments; they were operated on a second time and again allowed to be out of bed on the third day. In all three patients the retina became reattached in spite of two of the eyes being aphakic. The author suggests that these observations indicate that prolonged bed rest following surgery for retinal detachment is not always necessary.

Pereyra [196] reports that diathermy treatment of 180 patients with retinal detachment resulted in recovery in 81%. He states that one can expect the best results in (1) young patients with traumatic detachment of the retina, (2) nonmyopic patients, and (3) patients with only one tear or disinsertion in a recently detached retina.

Rosengren [197] reports 300 cases in which operation was performed for retinal detachment, in 234 (78%) of which cures were achieved. All except 16 of the

192. Gonzales, N. B.: Retinal Detachment in Binocular Ophthalmoscopy of the Periphery with Goldman's Contact Lens, Arch. Soc. oftal. hispano-am. **12**:386, 1952.

193. Dixon, J. M.: Binocular Indirect Ophthalmoscopes: The Trotterscope and the Heedscope, Tr. Am. Acad. Ophth. **56**:607, 1952.

194. Smith, R.: Suprachoroidal Air Injection for Detached Retina: Preliminary Report, Brit. J. Ophth. **36**:385, 1952.

195. Jervey, J. W.: Note on Postoperative Management of Retinal Detachment, A. M. A. Arch. Ophth. **47**:76, 1952.

196. Pereyra, L.: Clinical and Statistical Considerations on 230 Cases of Retinal Detachment, Gior. ital. oftal. **4**:356, 1951.

197. Rosengren, B.: Three Hundred Cases Operated upon for Retinal Detachment: Method and Results, Acta ophth. **30**:117, 1952.

patients had been followed for 12 to 96 months after operation. The technique of operation used consisted of surface diathermy plus puncture and injection of air in 276 cases, with resulting cures in 198 cases, and either diathermy or diathermy and puncture in 44 cases, in 36 of which cures were obtained.

Verrey [198] reports the results in 239 cases of retinal detachment treated surgically between 1944 and 1951 at the eye clinic of the University of Zurich. In 202 of these cases there was complete and permanent reattachment of the retinas; in 9.9% reattachment occurred but did not last, and in the remaining 28.2% the retina did not become reattached. The various causes for the failures are discussed and tabulated.

Gifford and Kauffman [199] report 9 successes in 14 cases of retinal detachment treated by surface coagulation with the Shahan thermaphore. Their criteria for success were complete reattachment, a full or nearly full visual field, and return to normal visual acuity or to the visual acuity the patient had before detachment. Subretinal fluid was released by a sclerotomy performed with a cataract knife.

An analysis of 1,084 cases of retinal detachment is made by Weve.[200] No hole could be found in the retina in 177 cases (13.3% of the total number), and apposition of the retina was achieved by diathermy in 50% of this group. Of 937 cases in which holes or tears were found, the retina was reattached in 70%. If cases of previous operation are excluded, the percentage of successes rises to 80. Two causes of operative failure are discussed. The first cause is the formation locally on the retina of a dense sheet of connective tissue. The second cause is the appearance of a new tear at the margin of the diathermy coagulation. Weve suggests that this type of tear is due to scleral shortening following diathermy coagulation and subsequent stretching as a result of intraocular pressure. The author treats this type of recurrence by his operation of "reefing" the sclera.

Weve [201] states that early recurrences of retinal detachments following surgery are caused either by star-shaped scars or by changes in the sclera. After diathermy coagulation the sclera shrinks; then, later, when the tension of the eye returns to normal, the sclera will stretch. When the stretching occurs in the retina also, new tears will appear. He states that these changes can be prevented by the use of scleral "reefing" or by x-ray therapy.

Philps [202] reports that 60 to 65% of cures in his clinic are the result of surgical reattachment of retinal detachments. He discusses the reasons for failure in the remaining cases under the following headings: (1) long-standing detachments, of six months' duration or more in which secondary changes have occurred; (2) detachments in which no hole can be found; (3) a massive vitreous retraction, so that the retina either breaks away again or never returns at all to its bed, (4) degeneration of the retina and vitreous accompanying high myopia, and (5) a violent postoperative reaction, ending in disturbances and hemorrhage in the vitreous.

198. Verrey F.: Critical Studies on Failures in Retinal Operations, Ophthalmologica **123**:271, 1952.

199. Gifford, E. S., Jr., and Kauffman, M. L.: Surface Therapy with the Shahan Thermaphore in Retinal Detachments, Am. J. Ophth. **35**:1201, 1952.

200. Weve, H.: Surgical Treatment of Retinal Detachment, Irish J. M. Sc. **6**:74, 1952.

201. Weve, H.: "Reefing" of the Sclera in Conjunction with Diathermy Coagulation of Retinal Holes, Docum. ophth. **56**:97, 1951.

202. Philps, A. S.: Modern Treatment of Retinal Detachment, Tr. Ophth. Soc. U. Kingdom **71**:631, 1951.

He advocates a preoperative period of bed rest with binocular bandages for all patients except those who have disinsertions in the lower half of the eye. If this is done, a fairly accurate forecast of the probable results of diathermy can be made at the end of the period of bed rest. He suggests the following guide for surgical treatment: (1) for oral disinsertion, diathermy; (2) if a retinal hole is found and there is some reposition with rest, diathermy; (3) if no hole is found, or if a hole is found but there is no improvement with rest, probably scleral resection.

Shipman [203] discusses 12 types of preoperative complications in retinal detachment, 12 types of complications encountered during operation, and 16 types of postoperative complications observed in 500 patients with retinal detachment.

Shapland [204] reports that from 1934 to the end of 1947 he had treated 146 eyes by diathermy, with 61% reattachments and 39% failures. From 1948 through 1950 he operated on 110 eyes, with reposition in 65%, improvement in 11.8% and failure in 22.7%. He suggests that the improvement in his results from 1948 through 1950 was due to the use of catholysis punctures at 2 ma., instead of the diathermy puncture and injection of air into the vitreous cavity used in most cases of superior detachment. He also discusses the results of scleral resection. A lamellar scleral resection was performed in 15 cases, with cure in 2, improvement in 9, failure in 3, and relapse in 1. A full-thickness scleral resection was done in four cases, with improvement in one and failure in three. In two cases a combined lamellar and full-thickness scleral resection was done with improvement in one and failure in one.

Lister [205] reports 120 cases in which scleral resections had been performed between July, 1947, and May, 1951. Lamellar resection was done in 18 instances, with cure in 2 cases (11%). Penetrating resections in 102 cases resulted in cure in 23 cases (22.5%). In 90 of the cases previous diathermy had been done; 17 (20%) of these were cured by a scleral resection. In 30 cases in which no previous surgery had been done, scleral resection resulted in a cure in 7 (23%). It is interesting to note that in 21 of these cases retinal detachments followed cataract extractions. Seventeen of the cataract extractions were extracapsular, and 4 were intracapsular. This ratio could not be explained by the fact that more extracapsular extractions were done at Moorfields, for from 1947 to 1951 intracapsular extractions had attained at least parity with extracapsular extractions.

Lister [206] notes that scleral resection has undergone a sudden revival in several countries almost simultaneously. He explains this by saying that while diathermy is preeminently successful in the majority of cases of retinal detachment, there is a sizable number of eyes in which this operation fails. Scleral resection is advocated by the author in the following types of cases: (1) detachments in aphakic eyes; (2) detachments in which no tear can be found; (3) detachment in which there are organized bands of vitreous; (4) detachments of long standing; (5) gross

203. Shipman, J. S.: Important Complications in Retinal Detachments and Their Influence on the Surgical Result, Am. J. Ophth. **35**:1263, 1952.

204. Shapland, C. D.: A Discussion of the Modern Treatment of Detachment of the Retina, Tr. Ophth. Soc. U. Kingdom **71**:651, 1951.

205. Lister, A.: Scleral Resection in the Treatment of Retinal Detachment, Tr. Ophth. Soc. Australia **11**:57, 1951.

206. Lister, A.: Discussion on Shapland.[204]

retinal degeneration, either senile or myopic, associated with multiple or very large tears, and (6) detachments in which diathermy has already failed. He describes in some detail his procedure for performing scleral resections. His report of the results of scleral resection at Moorfields between July, 1947, and July, 1951, is essentially the same as his previous one.[205]

The history of scleral resection is given in some detail by Shapland.[207] He then describes his technique for lamellar scleral resection. A strip of sclera, varying from 2 to 6 mm. in width, covering approximately one-half the globe, is outlined by an incision in the superficial sclera 2 to 3 mm. behind the rectus muscles. A suture is then passed through one tip of the sclera that is to be resected. The sclera is incised with a sharply ground cataract knife until the choroid can be seen through the few remaining fibers of sclera. The dissection is then carried out on this plane, a few fibers of the sclera being always left to protect the choroid from bulging into the wound. Silk sutures are inserted at intervals of 2.5 to 3 mm. as the strip of sclera is resected. The exposed deeper layers of the sclera and choroid are cauterized with 3% potassium hydroxide applied with a cotton swab. Subretinal fluid is released by a catholysis needle puncture through the choroid. The scleral sutures are tied. His results with this procedure were described above.[204]

Berliner[208] discusses the treatment of retinal detachment by scleral resection. His technique and the indications for operation are essentially the same as those previously described. His technique differs slightly from that used by Shapland. He begins his resection at the midportion of the scleral tongue and applies a ball diathermy cautery to the choroid, instead of potassium hydroxide.

Vesey[209] reports a patient with a large disinsertion in one eye. Resection of a strip of sclera 2 mm. wide and 20 mm. long without diathermy resulted in cure.

Paufique and his co-workers[210] describe their indications, techniques, and results in lamellar scleral resection plus diathermy in 57 eyes with a poor prognosis. Anatomical reattachment occurred in 31 cases, and failure resulted in 26, a percentage of successes of 54.38. A penetrating scleral resection without diathermy was done on seven eyes, and if these are eliminated the percentage of successes is raised to 60. The authors list the indications for the operation as recurrent retinal detachment, retinal detachment in high myopia, aphakia, arteriosclerotic degeneration or cataract accompanying the detachment, and multiple tears and severe cystic degeneration of the retina.

Hruby[211] advocates a combination of electrocoagulation and scleral shortening in cases of detachment with multiple retinal defects or shrinking foci in the retina itself. Of eight cases, anatomical healing was attained in seven with this type of operative procedure and in one reattachment was achieved by a second operation.

207. Shapland, C. D.: Scleral Resection—Lamellar, Tr. Ophth. Soc. U. Kingdom **71**:29, 1951.

208. Berliner, M. L.: Lamellar Resection of Sclera in Treatment of Retinal Detachment: Preliminary Report, A. M. A. Arch. Ophth. **48**:596, 1952.

209. Vesey, F. A.: Sclerectomy in Retinal Detachment with No Attempt to Seal the Disinsertion; Report of a Case Successfully Treated, Am. J. Ophth. **35**:708, 1952.

210. Paufique, L.; Hugonnier, R., and Moreau, P. G.: The Place of Scleral Resection in the Treatment of Detachment of the Retina: Indications, Personal Technique and Results, Ann. ocul. **185**:113, 1952.

211. Hruby, K.: Combined Operation in Detachment Cases with Very Poor Prognoses, Klin. Monatsbl. Augenh. **120**:481, 1952.

Sixteen cases of detachment in the temporal portion of the retina, including the macular area, are reported by Castroviejo and Torres-Lucena.[212] In all these cases the prognosis was poor. Scleral resection with diathermy coagulation was done, and in eight of the cases the retina became reattached, with restoration of visual field and improvement in visual acuity.

Fischer and von Nordheim[213] determined the sugar content of the subretinal fluid and of the blood in patients with retinal detachment. They found that when the quotient $\frac{\text{sugar concentration subretinal fluid}}{\text{sugar concentration blood}}$ equaled a value of less than 0.59, reattachment of the retina resulted in 24 patients, with a definite increase in vision. Four patients obtained anatomical healing but no improvement in vision, and all showed a quotient greater than 0.59. From their findings, they theorized that the sugar content in the retinal fluid is an indication of the functional state of the retina. An exception to this finding was noted in 12 patients with disinsertion at the ora serrata.

In an instructive article, Fischer[214] reviews the literature and discusses the formation of retinal cysts and cystoid degeneration of the retina. He divides the history of our knowledge into three periods. During the first, from 1827 to 1915, cysts were examined only anatomically and histologically. Toward the end of this period, cyst formation in the fovea and the ora serrata was considered to be the result of deficient vascularization. The fact that the temporal half of the ora serrata was most frequently affected was explained by its greater distance from the central retinal vessels. The second stage extended from 1915 to 1935. During this period the cause and manner of the development of the cyst remained obscure. However, it was noted that the formation of cysts and retinoschisis was closely related to retinal detachment. The experimental, and third, stage is not yet concluded.

The author demonstrates that when freshly removed retinas were treated before fixation in solutions of acids, bases, and salts of varying pH, there occurred a difference in the swelling capacity of the various layers of the retina. The rods and cones, the nerve fiber layer, and the plexiform layers become swollen very easily. Glial elements have less tendency to swell, and no swelling occurred in the inner and outer nuclear layers. The most susceptible region was the outer plexiform layer, which swells so very rapidly that retinoschisis usually appears in specimens before fixation.

Although these experiments are still in a preliminary stage, the author thinks that they suggest a possible mechanism of cyst formation. Capillary perisclerosis has been found in the retinas in some cases of retinal detachment. The tissue fluid originating in the choriocapillaris and nourishing the neuroepithelium is in perisclerosis of the peripheral vessels inadequately removed as a result of the obliteration of the perivascular fluid space, thus causing, by local accumulation, cyst-like cavities in the retina.

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212. Castroviejo, R., and Torres-Lucena, M.: Scleral Resection in the Treatment of Retinal Detachment, Arch. Soc. oftal. hispano-am. **12**:152, 1952.

213. Fischer, F. P., and von Nordheim, R. W.: Sugar Content of the Subretinal Fluid in Retinal Detachment, Docum. ophth. **5–6**:88, 1951.

214. Fischer, F. P.: Retinal Cysts and Cystoid Degeneration of the Retina, Docum. ophth. **5-6**:12, 1951.

O'Day [215] describes the histological examination of the eye of a patient who had hereditary retinal detachment. This patient's clinical history had previously been reported by Hamilton.[216] He was first observed in 1935, at the age of 12. His vision was reduced to light perception, and a hypermature cataract was present. The eye was enucleated 17 years later. Histological examination revealed a complete retinal detachment of the umbrella type. A dislocated, cataractous lens was noted in the anterior chamber. There was widespread cystic degeneration of the retina. In some areas the retinal vessels were surrounded by collections of round cells, and there was evidence of retinitis proliferans. O'Day goes on to describe cystic degeneration in other patients; this is most commonly found in the outer plexiform layer and the nuclear layer. He concludes that the eye of the patient reported in this study showed the end-results of fetal retinitis.

Experimental permanent retinal detachments are extremely difficult to produce in rabbits without destroying the globe. Robertson and Shirley [217] used a technique described by Castroviejo in which a cataract knife is plunged into the vitreous to produce a retinal tear. Half the vitreous is then withdrawn through a blunt needle. The retina is detached from the choroid by passing an iris repositor between these two layers, and the vitreous is injected between the retina and the choroid.

Using animals in which a satisfactory detachment of the retina had been obtained, they attempted to produce a reattachment by the injection of physiological glue into the subretinal fluid. The glue was a mixture of concentrated plasma, 1 part, to thrombin, 3 parts. A severe chorioretinitis followed, and four eyes became phthisical.

Histological examination showed that the retinas of three control animals were not reattached. The choroid and retina appeared healthy. All eyes in which the physiological glue was used showed severe chorioretinitis, with almost complete destruction of the retina.

TRAUMA

The ocular effects of methyl alcohol poisoning were described by Benton and Calhoun.[218] A series of 320 patients who consumed bootleg whiskey containing 35% methyl alcohol and 15% ethyl alcohol was studied. Some of the patients consumed as little as 90 cc., and others as much as 750 cc., of the alcoholic mixture. Symptoms usually developed within 18 to 48 hours after ingestion of the methyl alcohol, but in a few instances transitory symptoms were noted within one hour. The patients complained of visual disturbances, weakness, abdominal pain, nausea, vomiting, headache, dizziness, and shortness of breath. The initial visual symptoms ranged from spots before the eyes to complete blindness.

The most important guide to diagnosis, therapy, and prognosis in patients poisoned with methyl alcohol is the carbon dioxide-combining capacity of the blood. When this was normal (above 26 mEq. per liter), none of the 181 patients were treated. A follow-up study was done on 94 (or 50%) of this group two to six

215. O'Day, K.: Retinal Cysts, Tr. Ophth. Soc. Australia **11**:90, 1951.

216. Hamilton, J. B.: Inherited Retinal Detachments, Tr. Ophth. Soc. Australia **6**:113, 1946.

217. Robertson, R. W., and Shirley, S. Y.: Experimental Retinal Detachments and the Use of Physiologic Glue in Rabbits' Eyes, Am. J. Ophth. **35**:565, 1952.

218. Benton, C. D., Jr., and Calhoun, F. P., Jr.: Ocular Effects of Methyl Alcohol Poisoning: Report of a Catastrophe Involving 320 Persons, Tr. Am. Acad. Ophth. **56**:875, 1952.

months after the poisoning and not one patient had evidence of visual damage. The carbon dioxide-combining power of the blood was 25 mEq. per liter or below in the remaining 132 patients, 37 of whom died.

The absence of the pupillary light reflex is a poor prognostic sign for both visual acuity and life. The visible retinal changes which result from the poisoning are hyperemia of the optic nerve head and superficial edema of the papilla and surrounding retina, followed by simple atrophy of the optic nerve. All patients with severe retinal edema had some degree of permanent visual loss. The earliest central field defect was usually a cecocentral scotoma. Later, more complicated types of visual field defects appeared. Partial or complete recovery of the initially reduced vision was noted in most patients. If visual acuity did not return to normal within six days after treatment was begun, it invariably dropped again to a very low level. The final vision was 20/200 in each eye of six patients and in one eye of three patients.

The most effective treatment of acute methyl alcohol poisoning is intensive and prolonged alkalization.

Potts, in a discussion on this paper, points out that loss of vision in methyl alcohol poisoning is not due to acidosis alone. It is true in this series that visual impairment was usually associated with acidosis, but the degree of visual damage did not exactly parallel the degree of acidosis. Likewise, he states that if acidosis alone was the cause of the visual loss, such changes should occur in patients with diabetic acidosis. He thinks that a basic toxic mechanism is present in methyl alcohol poisoning which produces both the retinal changes and the acidosis but that no cause-and-effect relationship exists between the two.

Löhlein [219] describes the case of a woman aged 35 who received 1.2 gm. of ethyl hydrocupreine hydrochloride (Optochin) in 24 hours (adult dose, 1.5 gm. in 24 hours) for pneumonia when she was one year of age. One day after administration the pupils were wide and did not react to light. The visual acuity improved after the 11th day. By the age of 6 months she had nystagmus, pale optic disks, and narrowed retinal blood vessels. At the age of 35 her visual acuity was 1/6 in each eye. The visual fields were constricted, with a paracentral scotoma. She complained of night blindness. Ophthalmoscopic examination revealed narrowing of the vessels and pale optic disks, very similar to the picture found in retinitis pigmentosa. The patient was married and had four normal children. The similarity of the findings to those in retinitis pigmentosa is discussed.

Hogan [220] discusses ocular contusion injuries and notes the presence of macular edema (commotio retinae), retinal hemorrhages, and disinsertions of the retina.

Two cases of retinal edema and hemorrhage following trauma to the chest and abdomen with multiple bone fractures are described by Hryniewska.[221] The author favors the theory that these retinal changes are due to fat emboli. Tanaka [222] describes similar findings in an 11-year-old girl who was run over by a motorcycle.

219. Löhlein, W.: Results of Heavy Optochin Poisoning in Childhood on the Retina and Optic Nerve, Klin. Monatsbl. Augenh. **121**:25, 1952.

220. Hogan, M. J.: Ocular Contusions, Am. J. Ophth. **35**:1115, 1952.

221. Hryniewska, H.: Angiopathia Retinae Traumatica, Klin. oczna **22**:31, 1952.

222. Tanaka, T.: A Case of Traumatic Retinal Angiopathy (Purtscher's Disease), Shikoku Acta med. **2**:21, 1951.

Klimes and Eröss [223] state that the initial reaction of the fundus following electric shock is a constriction of the vessels of the fundus and an increased diastolic pressure in the central retinal artery, both as a result of stimulation of the sympathetic nervous system. Later symptoms are due to parasympathetic action, which causes dilatation of the retinal vessels and a decrease in the retinal blood pressure.

Klemens [224] describes the case of a woman aged 41 who had multiple small cysts throughout the fundus as a result of shock from a high-tension electric current.

Ten cases of retinal injury which resulted from observation of a solar eclipse without proper protection of the eyes are described by Gotthelf.[225] Most of the patients had been examined by the author before the injury occurred. The fundus changes consisted mostly of minute yellowish-white spots in the macular area or a dark-red circular area in the fovea, causing a central scotoma.

Favory and Sédan [226] describe the ocular injuries which may result from boxing. Retinal hemorrhages in the far periphery of the fundus are frequent. These may lead to formation of tears and retinal detachment. Berlin's traumatic edema of the retina is a frequent occurrence but does not leave sequelae in most instances.

Siebeck [227] states that retinal lesions following blunt injuries to the globe are caused by angiospasm. He describes two patients who were shot in an eye with an air pistol. The retinal vessels were strikingly narrowed in the area of the contusion.

Roper-Hall [228] analyzed 200 cases of intraocular foreign bodies occurring in Birmingham, England, from 1946 to 1950 inclusive. He states that diathermy coagulation of the sclera, either in the area where the foreign body penetrated the sclera or in the area where it was extracted, was not necessary to prevent retinal detachments.

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223. Klimes, K., and Eröss, S.: Aspects of the Fundus in Cases of Electrical Shock, Magy. Bel. Arch. **5**:94, 1952.

224. Klemens, F.: Rare Changes in the Fundus: Problem of Injury by High Tension Current, Klin. Monatsbl. Augenh. **120**:90, 1952.

225. Gotthelf, E.: Eye Injuries After an Eclipse of the Sun, Acta med. orient. **11**:103, 1952.

226. Favory, A., and Sédan, J.: Eye Injuries in Boxing, Arch. opht. **11**:429, 1951.

227. Siebeck, R.: Traumatic Lesions of the Retinal Vessels in Juvenile Hypertension, Klin. Monatsbl. Augenh. **121**:46, 1952.

228. Roper-Hall, M. J.: Treatment of Intraocular Foreign Bodies with Results and Complications, with Special Reference to Detachment of the Retina, Ann. ocul. **185**:468, 1952.

News and Comment

GENERAL NEWS

Wills Eye Hospital: Fifth Annual Clinical Conference.—The fifth annual clinical conference of the staff and ex-residents of the Wills Eye Hospital, Philadelphia, was held at the hospital on March 20 and 21, 1953. The registration approximated 350.

The meeting was opened by Dr. Ivan J. Koenig with a clinicopathologic case report of a malignant melanoma of the iris in a one-eyed patient, under observation for 20 years.

Dr. Clyde H. Jacobs presented a moving picture showing several cataract extractions performed with intravenous thiopental (Pentothal) sodium anesthesia.

The hospital's research staff, under the direction of Dr. Irving H. Leopold, made the following reports on five of their current projects:

1. Drs. Virginia L. Weimer, Harry Green, and Irving H. Leopold described a method for determining steroid concentration in the intraocular fluids which has enabled them to establish the presence of certain steroids in these fluids of the experimental animal eye.

2. Drs. J. Eisenberg, Irving H. Leopold, and J. W. Hallett presented evidence showing that the radioactive isotope P^{32} could be helpful in the differential diagnosis of intraocular tumors. The ability of the normal eye to concentrate P^{32} was determined, and the percentage variation of over 50 normal eyes was used for this determination. More than 40 eyes suspected of having intraocular neoplasms were then subjected to this test. Whenever the probe could reach the suspected area, positive readings were obtained in histologically confirmed cases of intraocular neoplasms. In cases of neoplasms in the extreme posterior segment of the globe, where it was difficult for the probe to reach, negative results were occasionally produced. In all cases of serous detachment the results were negative. Five inflammatory conditions gave positive uptake values. The authors tentatively concluded that this is a valuable aid in differential diagnosis. They offered it simply as an adjunct, and not as a method to supplant any techniques already available.

3. Drs. William F. Kratka and Irving H. Leopold arrived at the following tentative conclusions from their studies with isonicotinic acid hydrazides in experimental and clinical ocular tuberculosis:

(*a*) Isonicotinic hydrazides will control experimental and clinical ocular tuberculosis when therapy is maintained for a long period. In the experimental animal improvement was not noted until after 50 days of therapy, when treatment was instituted 9 days after inoculation into the anterior chamber or cornea.

(*b*) If therapy was stopped too soon, even though there was obvious clinical improvement, there might be recurrences. The best results were obtained in cases in which therapy was continued for six months. Evidence of clinical quiescence of the lesion often belies the true picture, in that histologic preparations of the same eyes show definite activity, which may account for the numerous recurrences with too short a period of therapy.

(*c*) Therapeutic results in experimental lesions compared favorably with those of streptomycin and paraaminosalicylic acid. In some instances, the results obtained with the combination of the three drugs were superior to those with streptomycin

or paraaminosalicylic acid alone. In some cases the results with isonicotinic acid in the experimental eye were superior to those obtained with streptomycin or paraaminosalicylic acid.

(*d*) Pentration studies were also made after the subconjunctival, intramuscular, and topical administration of isonicotinic hydrazides. With all forms of administration adequate penetration into the intraocular tissues and fluids could be obtained.

4. Drs. J. O'Rourke and Irving H. Leopold reported the inability of the enzymes streptodornase and streptokinase to lyse rabbit blood, and they further demonstrated the necessity of a human-serum factor, namely, plasminogen, for the lysis of human and experimental-animal blood by these enzymes. Their studies indicated a safe dose of these enzyme preparations for intracameral injection. It was shown that much of the toxicity of the commercially available enzyme streptokinase is due to the contained hyaluronidase. The action of these fibrolytic enzymes is greatly influenced by changes in temperature, more so than by concentration. The length of time that the enzyme remains in the anterior chamber is of practical importance. In trials against standard experimentally induced hemorrhages in the anterior chamber, injection of the enzymes combined with paracentesis and flushing of the anterior chamber gave the most satisfactory results. In low concentrations, these enzymes lose their anticoagulant properties. Apparently, this property may be due to impurities. These enzymes were found to have the ability of provoking a response to antibiotics.

5. Drs. Adolph W. Vogel and Irving H. Leopold described a method for producing experimental lesions of the posterior segment. They demonstrated the production of localized inflammatory and neoplastic lesions in the choroid with their technique.

Several other papers were presented. Drs. Warren S. Reese and Turgut Hamdi reported their experiences with the Ridley implant in cataract surgery. Dr. Reese had performed 14 of these operations at the hospital during the previous year, with favorable results in 12.

Dr. P. Robb McDonald spoke on the indications for scleral resection in retinal separation as a primary procedure, rather than as a "last-resort," in cases in which the prognosis is poor.

Drs. Wilfred E. Fry, Max Kasser, and Samuel DeLong advocated excision of partial thickness of the sclera as a valuable addition to the surgical treatment of retinal detachment. Their conclusions were based on experimental work on rabbit eyes.

Dr. Robert E. Shoemaker discussed the ocular complications of erythema multiforme exudativum. He described an unusual case of an 8-year-old boy who presented symptoms of an eruptive fever, stomatitis, endophthalmitis with reduction of vision to light perception, and arthritis with effusion. This he considered a probable variant of erythema multiforme. With the systemic administration of cortisone (Cortone) all symptoms disappeared within a week, except for the eyes. Vision eventually recovered to 20/30 three months after the onset.

Dr. Raynold N. Berke accompanied his splendid moving picture on resection of the levator palpebrae muscle for blepharoptosis with many practical and instructive comments.

In general terms, Dr. Edmund B. Spaeth gave the indications and basic principles for surgical correction of the vertically acting muscles in strabismus, his conclusions being based on more than 800 cases occurring over a three-year period.

The Arthur J. Bedell Lecture was given by Dr. Ellen F. Regan in the absence of Dr. John H. Dunnington. The paper was entitled "Absorbable Sutures in Cataract Surgery," and Dr. Dunnington and Dr. Regan were joint authors. Their conclusions were based on experimental studies and clinical observations. They found that the initial response with absorable surgical (gut) sutures U. S. P. was a

polymorphonuclear-leucocyte reaction, which with the plain type was rapid and pronounced, leading to early disintegration of the suture. When mildly chromicized material was used, this polymorphonuclear-cell response was less pronounced, and the suture remained intact about seven days. Fibroblastic proliferation became evident about the fifth day, as did increased vascularization of the wound. During the 2d week the healing process progressed, and at the end of 14 days the wounds appeared well healed, but somewhat broader and more vascularized than comparable ones closed with nonabsorbable surgical (silk) sutures. The increased vascularization of these wounds is an undesirable feature, but this is counterbalanced by the relative absence of tissue necrosis and of epithelial invasion, which the authors have found in the presence of silk sutures. In view of these findings and their present clinical experience, the authors felt that mildly chromicized absorbable surgical suture material is satisfactory for closing cataract incisions.

An important "repeat" feature of the conference was the color-television program, presented by the members of the surgical staff of the hospital, through the courtesy of Smith, Kline & French Laboratories. These surgeons are becoming expert TV operators, as well as commentators.

Fourteen commercial exhibitors were also on hand with splendid displays.

As in former years, the meeting was not without its social side. The first evening there was an informal reception for the doctors and their wives at the Barclay Hotel, followed by a delicious dinner. On Saturday evening the Wills Eye Hospital Society held its annual dinner at the Union League Club.

Books

Table of Tests for Color Blindness. By Karl Velhagen. Price, 18.40 German marks. Georg Thieme, Thomaskirchhof 21, (10b) Leipzig C. 1 (Russian Zone), 1952.

This is a series of plates similar to the Ishihara plates for testing different degrees and kinds of color blindness.

Basedow's Disease. By H. Sattler; English translation by G. W. and J. F. Marchand. Price, $10. Pp. 605. Grune & Stratton, Inc., 381 4th Ave., New York 16, 1952.

This book is of great historical interest. Its author, who was one of the world's best-known ophthalmologists, died in 1929, at the age of 85. He collected the literature on Graves's disease, or, as the Germans prefer to call it, Basedow's disease, covering the period from 1722 to 1909. It will be extremely valuable to all those who wish an exhaustive treatise on this disease, and of special interest to ophthalmologists concerned with the ocular manifestations of Graves's disease.

Introduction to Physiological Optics. Second edition. By Armin von Tschermak-Seysenegg, translated by Paul Boeder, Ph.D. Price, $10.50, Pp. 305, with 151 illustrations. Charles C Thomas, Publisher, Springfield, Ill., 1952.

For those who have struggled to read Tschermak's "Introduction to Physiologic Optics" in the original German, this excellent translation by Dr. Boeder will prove a boon indeed. As Dr. Burian points out in the preface, few men are as intimately acquainted with the subject as Dr. Boeder, and only "those who are able to study Tschermak's German text will fully appreciate the difficulties which the translator had to overcome in order to present us with such a clear, fluent English text while nowhere either adding to or subtracting from the original." The translation of this classic was authorized by the American Committee on Optics and Visual Physiology of the Section on Ophthalmology of the American Medical Association, the American Ophthalmological Society, and the American Academy of Ophthalmology and Otolaryngology, who considered the text unique in its field.

Even with the expert treatment Dr. Boeder has given it, this book is in no sense an "introduction" to the subject matter. The title is also misleading if one interprets physiological optics to be the application of geometrical optics to the eye. Only the first chapter treats, in a very brief manner, the optical image, and in twenty-eight pages it covers image formation, the aberrations of the eye and the physiological correction of these aberrations, irradiation, and the problems of visual acuity. The rest of the book deals with the psychophysiology of vision.

Chapter II considers the light sense; Chapter III, the color sense. Then follow two chapters on uniocular and binocular space sense, and the final one, Chapter VI, deals with the physiology of ocular motility.

Only those well founded in basic psychophysiological concepts could read these chapters and come away feeling rewarded. Even the last chapter constitutes a postgraduate course in ocular motility. From every point of view, therefore, the book can be recommended only to those well versed in the subject, but for these it is worth considerable study.

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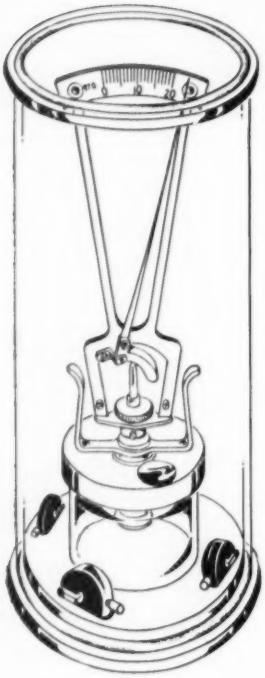

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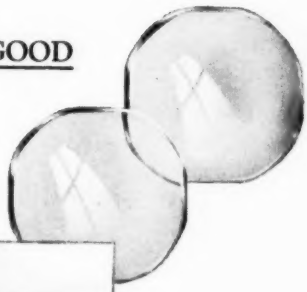

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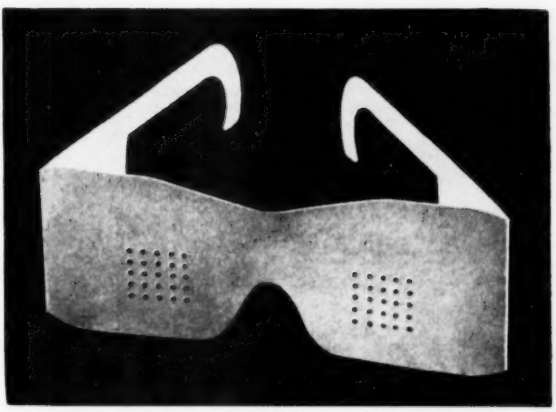

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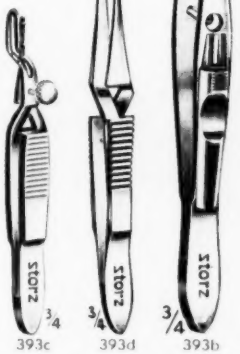

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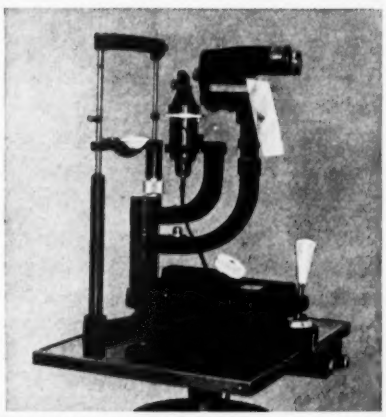

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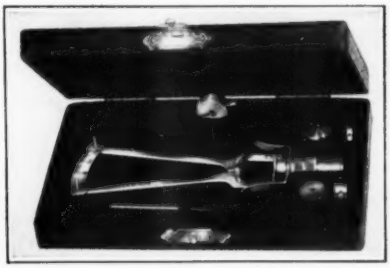

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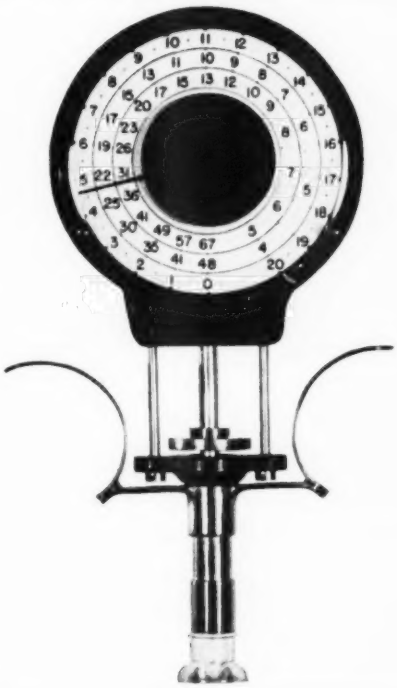

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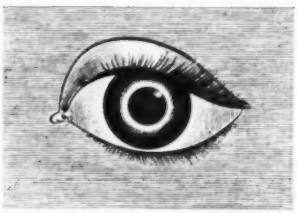

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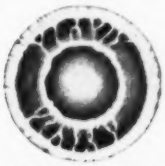

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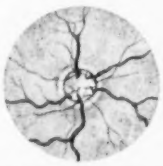

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